

The Effects of a Psychosocial Behavioural Intervention on Arterial Health in Children

by

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**Abstract**

This study evaluated the effects of a Leisure and Well-Being Model (LWM) psychosocial intervention on arterial health, measured by arterial stiffness and thickness, in 82 children aged 10-13 (n=41; intervention, n=41; control) over one year. The intervention was to provide children with the awareness, skill development, and application of positive emotion, personal strengths, coping, and free-time vitality. Results showed no change in arterial health for children exposed to the intervention compared to controls. However, a significant systolic blood pressure decrease was found in children exposed to the intervention and increased in those of the control group ( $F(1, 73) = 4.085$ ,  $p = 0.047$ ). This is the first study to show that a psychosocial intervention has a positive effect on childhood cardiovascular health within one year. Hence, if exposed for-or followed for- a longer period of time, it may be possible to see further improvements in arterial health.

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## List of Abbreviations

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AIx	Augmentation Index
aPHV	Age from peak height velocity
BMI	Body mass index
BP	Blood pressure
CCA	Common carotid artery
CSA	Cross-sectional area
CVD	Cardiovascular disease
CW	Continuous wave
DBP	Diastolic blood pressure
dCSA	Diastolic cross-sectional area
HDL	High-density lipoproteins
HR	Heart rate
HT	Hypertensive
Hz	Hertz
IMT	Intima-media thickness
kg	Kilograms
KHz	Kilohertz
LDL	Low-density lipoproteins
LWM	Leisure and Well-Being Model
MHz	Megahertz
NO	Nitric oxide
NT	Normotensive
PA	Physical activity
P <sub>d</sub>	Diastolic pressure
PHV	Peak height velocity
PP	Pulse pressure
PreHT	Pre-hypertensive
P <sub>s</sub>	Systolic pressure
PW	Pulse wave
PWV	Pulse wave velocity
SBP	Systolic blood pressure
sCSA	Systolic cross-sectional area
SD	Standard deviation
SES	Socioeconomic status
WHR	Waist-to-hip ratio

## **Chapter 1.0 Introduction**

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### **1.1 Preamble**

Proper functioning of the arterial system relies on its own structure and different components of the human body such as hormones and the autonomic nervous system. Knowing how the arterial system functions under healthy conditions helps to understand related risk factors and causes of arterial disease. Large artery pathology is a major contributor of cardiovascular disease (CVD) being a leading cause of morbidity and mortality in developed countries (Arnett, Evans, & Riley, 1994). Understanding risk factors that influence the normal functioning of the arterial system allows for early detection and prevention of disease. There are several non-invasive measures of arterial function and structure that have been shown useful in predicting the development of CVD which include measures of intima-media thickness (IMT) and arterial stiffness (Arnett, Evans, & Riley, 1994; Blacher et al., 1999; Grobbee & Bots, 1994).

In adults, measures of arterial stiffness and carotid IMT have been found to be associated with traditional risk factors such as, hypertension, diabetes, obesity, and hyperlipidemia, as well as are associated with the development and progression of CVD (Riggio et al, 2010; Christen et al., 2010; Resnick et al., 1997). These same non-invasive measures of arterial health have also found that such traditional risk factors affect arterial health at a very young age, beginning in childhood (Berenson, Srinivasan, Bao, Newman, & et al, 1998; Banach et al., 2010; Iannuzzi et al., 2004; Litwin et al., 2004; Tounian et al., 2001; Urbina et al., 2009). Additionally, elevated arterial stiffness and IMT in children have been found to predict the development of CVD in adulthood (Juonala et al.,

2005; Li, Chen, Srinivasan, & Berenson, 2004; Raitakari et al., 2003). In response to these findings, many lifestyle interventions have been proposed for children in order to slow down the progression of CVD and/or prevent certain risk factors from developing. These lifestyle interventions focus on increasing physical activity (PA) and eating a well-balanced diet, both of which provide enough evidence to suggest their beneficial effects on arterial health (Woo et al., 2004; Meyer, Kundt, Lenschow, Schuff-Werner, & Kienast, 2006).

However, even with the implementation of lifestyle interventions, incidence of CVD risk factors is increasingly becoming a problem during childhood. Therefore, focusing only on specific lifestyle risk factors, such as lack of exercise and poor diet, cannot explain fully the development of arterial stiffness and thickness in children. There must be other factors which influence arterial health. These risk factors may occur further away on the causal chain of arterial disease, but still have a significant impact on arterial health. In fact, behavioural science literature has researched a number of different psychosocial factors that may be examples of these indirect risk factors. This research attempts to understand how or why people come to be exposed to more direct causes/risk factors of CVD, such as, obesity, diabetes, hyperlipidemia and hypertension, even with knowledge of the benefits behind lifestyle interventions. The literature has shown a significant association between psychosocial factors and different disease outcomes (Bruce et al., 2009; Everson-Rose & Lewis, 2005; Strike & Steptoe, 2004), including improving quality of life of patients with disease and influencing biological processes thought to enhance disease progression (Bruce et al., 2009; Everson-Rose & Lewis, 2005). However, research that has been done looks mostly at the effects of psychosocial



interventions as a therapy or treatment strategy to increase longevity and slow progression of disease that has already developed in adults (Sullivan et al., 2009); while studies looking at how children may benefit earlier on to prevent such exposure are not found. Psychosocial factors as a cause of arterial disease tend to be overlooked because they are not direct causes and so research in children is limited. If such a psychosocial intervention was examined in children, it could explain how a person's life circumstances and behaviour can shape their exposure to more direct causes of arterial disease, and that these behaviours could be changed to increase arterial health and slow the progression of CVD.

Focusing on a psychosocial intervention in children encourages a shift in research from looking at preventive factors that are most direct to ones further down the causal pathway. This shift in focus is occurring in other areas of health as well. The profession of therapeutic recreation has introduced a theoretical model, The Leisure and Well-Being Model (LWM) that could provide an appropriate basis for the development of a psychosocial intervention to address cardiovascular health in children (Carruthers & Hood, 2007). The main outcome goal of the LWM is to increase individual well-being. Having this endpoint in mind allows for the examination of other factors that may contribute to the attainment of this end goal. Therefore, to assist one along the pathway towards well-being, the two main components selected to be integrated into this LWM include: 1) experiencing positive affect, and emotion on a daily basis, and 2) the cultivation and expression of one's full potential –including strengths, capacities, and assets (Hood & Carruthers, 2007). The model further explains that reaching one's full potential and increasing positive emotion can be achieved by certain methods of coping

and maximizing leisure experiences (Hood & Carruthers, 2007). Using this framework as the basis of a psychosocial intervention would allow for a new way to look at the effects of psychosocial factors on children's arterial health. During developmental stages, such an intervention may be a powerful strategy for prevention of disease development and/or progression. Therefore, the psychosocial intervention implemented will provide awareness, application and skill development of positive emotion, personal strengths, coping, and free-time vitality. The outcome goal is to increase arterial health in children whom may already be at CVD risk and to help prevent further risk factor exposure. Such an intervention is needed to start looking at psychosocial factors more seriously when it comes to bettering child health.

## **1.2 Objective**

The aim of this study was to investigate the effects of the LWM psychosocial intervention on arterial health, defined by the measure of arterial stiffness and thickness, in children and adolescents. This psychosocial intervention will provide children with awareness, skill development, and application of positive emotion, personal strengths, coping, and free-time vitality.

## **1.3 Hypothesis**

It was hypothesized that children who were exposed to the psychosocial intervention would have increased arterial health compared to those children who were not exposed to the intervention.

## **Chapter 2.0 Literature Review: Arterial Health**

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### **2.1 The Arterial System**

To be able to understand the effects of any intervention on measures of arterial health, one must first understand the normal functions and structure of the arterial system. As an introduction, this system consists of a network of elastic conduits and high-resistance vessels, which carry blood to and from all areas of the body. The aorta is the largest vessel and is responsible for pushing oxygenated blood throughout the systemic arterial system, while the respiratory system is supplied by the pulmonary arteries (Wilson, 2006). Travelling farther from the heart, the aorta and pulmonary arteries branch out, eventually forming arterioles which regulate the distribution of blood flow to the capillaries. Distributing blood to the capillaries allows for the supply of oxygen and nutrients necessary for bodily tissues to function (Berne & Levy, 1997). A traditional model of the arterial system is based on a Windkessel, an old-fashioned fire engine hose (or a hydraulic filter). Where this Windkessel is able to change pulsatile flow from a pump into a stream-like flow through the hose nozzle, the arterial system can convert intermittent output from the heart to a steady flow in the periphery (O'Rourke, Staessen, Vlachopoulos, Duprez, & Plante, 2002). The heart fills with and ejects blood in alternating cycles termed respectfully as diastole and systole, this cyclic behaviour is what creates the intermittent flow. Under normal conditions during systole, approximately 40% of stroke volume is forwarded directly to peripheral tissues (kinetic energy) and the remainder is stored as potential energy retained by stretching of the capacitance arteries (Berne & Levy, 1997). During diastole, the potential energy is converted into peripheral run-off by arterial recoiling, allowing constant flow through the

capillaries. If the vessels were to become stiff, most of the blood pumped from the left ventricle would be immediately converted to flow, resulting in reduced peripheral run-off during diastole. These changes lead to an increased load on the left ventricle and reduced coronary-flow, thereby increasing the risk of CVD events (Berne & Levy, 1997). This is a brief introduction into the functioning of the arterial system but a more detailed review of the physiology will provide the background knowledge needed to understand measures of arterial health able to quantify effects of an intervention on the arterial system.

### ***2.1.1 Arterial Physiology***

An artery is comprised of three layers; the intima or tunica interna (inner most layer), the tunica media (middle layer), and the tunica externa or adventitia (outer most layer). The intima consists of a layer of endothelial cells which have been shown to elicit contraction and relaxation of the vascular smooth muscles, as well as a number of metabolic and endocrine functions playing a critical role in disease states (Waller et al., 1992). The tunica media is the thickest layer composed mainly of smooth muscle cells and connective tissue (collagen and elastin); responsible for the contractile and elastic properties of the vessel. In the central arteries, elastin is the dominant component, while travelling further into the periphery collagen predominates (Safar, Levy & Struijker Boudier, 2003). The abundance of elastin in the central arteries enables them to withstand and filter out large pressure fluctuations from the pumping action of the ventricles. Collagen is more firm than elastin which means that the further away the artery is from the heart, the stiffer the wall (Nichols & O'Rourke, 2005). The arterial system's elastin-to-collagen ratios are normally held relatively stable by the existing balance between production and degradation. However, the stimulation of inflammatory processes,

increased blood pressure (BP), and aging may all lead to the overproduction of collagen and diminished amounts of elastin contributing to arterial stiffness (Zieman, Melenovsky, & Kass, 2005). In addition to the differing elastin-to-collagen ratios between the central and peripheral arteries, peripheral arteries also have the thickest tunica media layer of vascular smooth muscle cells. Vascular smooth muscle cells respond to incoming signals originating from various sources including the endothelium, humoral stimuli, and the autonomic nervous system causing either vasoconstriction or vasodilation depending on metabolic demands and myogenic responses (Berne & Levy, 1997). Since small changes in blood vessel diameter greatly influence blood flow and BP, the activities of the tunica media are critical in regulating circulatory dynamics (Nichols & O'Rourke, 2005; Waller et al., 1992).

As stated above, the intima layer of the arterial wall consists of endothelial cells. These cells locally release a number of different vasoactive substances of which the primary vasodilator is nitric oxide (NO). Also known as endothelium-derived relaxing factor, NO has a major influence on vascular smooth muscle tone and BP and, thus, also plays a role in the regulation of arterial stiffness (Wilkinson, Franklin, & Cockcroft, 2004). This vasoactive substance is derived through a pathway that generates continual release of NO, maintaining BP and attenuating sympathetic vasoconstriction. The NO pathway can also be enhanced by such substances as acetylcholine, bradykinin, and shear stress (Harris & Matthews, 2004). A brief review by Wilkinson and colleagues (2004) states that endothelial dysfunction, characterized by a decreased bioavailability of NO, is a predictor of cardiovascular risk (Wilkinson, Franklin, & Cockcroft, 2004). Wilkinson et al. (2004) further communicate that conditions associated with endothelial dysfunction,

such as hypercholesterolemia and diabetes, are also associated with increased arterial stiffness. Additionally, interventions that improve endothelial function also reduced stiffness, suggesting that NO may itself regulate arterial stiffness (Wilkinson, Franklin, & Cockcroft, 2004). The endothelium also releases contracting factors such as endothelin-1 and prostaglandin H<sub>2</sub>. Stimuli that release these factors include norepinephrine, thrombin, and luminal stretching (Harris & Matthews, 2004). In a healthy endothelium, NO can inhibit excessive release of endothelin-1 from the endothelium however in some disease states, such as hypertension, higher levels of endothelin-1 have been reported (Harris & Matthews, 2004).

The endothelium and the autonomic nervous system work together to maintain vascular smooth muscle tone. The balance between the two allows for appropriate vessel tone and function, thus, the regulation of arterial stiffness. Although endothelial cells in the periphery may receive direct neural innervations from the autonomic nervous system, cells along the central vessels do not. Therefore, neurotransmitter influence on endothelial function must be exerted by circulating levels or by diffusion through the smooth muscle cell layer (Harris & Matthews, 2004). The extent to which the autonomic nervous system controls vessel tone is also influenced by the endothelium. The release of NO decreases the sensitivity of smooth muscle cells to the vasoconstrictor effects of the sympathetic nervous system activity. Simultaneously NO increases the central and peripheral response to parasympathetic nervous system activity by enhancing the sensitivity of sites of action (Harris & Matthews, 2004).

In addition to understanding normal arterial function and structure, the above section explains other components of the human body that affect normal functioning of

the arterial system. This will be helpful later in the discussion of risk factors and to what degree they disrupt normal cardiovascular functioning. It also will be helpful now leading into the discussion of arterial health measures which are used to diagnose and/or predict arterial disease development, and thereby also quantify the effects of preventative interventions on the arterial system.

## **2.2 Arterial Health Measures**

CVD is a leading cause of morbidity and mortality in developed countries, with a major contributor being large artery pathology (Arnett, Evans, & Riley, 1994). Although CVD outcomes are almost entirely restricted to adults, a great amount of evidence has demonstrated that the presence of risk factors during childhood could lead to harmful changes in arterial structure and function in youth. In fact, fatty streaks have been identified in the intima of large arteries of children and adolescence as young as 2 years old (Berenson, Srinivasan, Bao, Newman, & et al, 1998). The ability to evaluate arterial function and structure before further complications and progression into adulthood is an important aspect of early detection and intervention. Although there are several non-invasive measures of arterial function and structure that have been shown as useful in predicting the development of CVD, measures of intima-media thickness and arterial stiffness have emerged as very powerful predictors.

Increased arterial stiffness has been found to be correlated with an increased risk of cardiovascular events, and to be one of the most important determinants of increased systolic pressure (SBP) and pulse pressure (PP) in aging populations (O'Rourke, Staessen, Vlachopoulos, Duprez, & Plante, 2002). Arterial stiffness is one of the earliest

detectable manifestations of adverse structural and functional changes within the arterial wall found to be correlated with the presence of atherosclerosis (Arnett, Evans, & Riley, 1994; Blacher et al., 1999). Also, it appears to be a modifiable measure sensitive to changes in lifestyle factors (Tanaka & Safar, 2005). In terms of measuring arterial stiffness, O'Rourke and Mancia (1999) state that it is important to consider that not all arteries are homogenous when quantifying arterial stiffness. Stiffness can be different within the same artery, between different arteries and at different pressures (O'Rourke & Mancia, 1999). In the literature, there are several different terms used to explain the elastic properties of the arterial wall which can lead to confusion and complication when trying to compare between studies. The umbrella term mostly used is arterial stiffness with various indices quantifying it. These indices include: augmentation index (AIx), elastic modulus, stiffness index, pulse wave velocity (PWV), compliance, and distensibility. No one measure has proved superior but this thesis will focus on the most common measures: PWV, compliance, and distensibility. IMT is another measure that this thesis will focus on and has also been shown to be a strong marker of structural and functional vessel wall properties, allowing early evaluation of arterial health (Grobbe & Bots, 1994).

### ***2.2.1 Arterial Stiffness Indices***

#### *2.2.1.1 Pulse Wave Velocity*

As the heart pumps, blood is ejected from the left ventricle and an arterial pressure wave (incident wave) is generated that travels along the vascular tree, and is reflected back at different points- mainly sites of increased impedance and/or branching



at resistant arterioles (Schiffrin, 2004). As the reflected waves travel back, they interact with the incident waves which distort them giving a final amplitude and shape of a measured pressure wave. The shape of each pressure wave and the time of interaction between both incident and reflected waves depend on a number of factors. The interaction can occur at different phases of the cardiac cycle depending on the traveling distance and the speed of the pressure waves. Also, differing elastic properties throughout the vascular tree can change the shape of the arterial waveform (Schiffrin, 2004). The high elasticity of the central arteries is the result of a high elastin-to-collagen ratio in their walls, which progressively decreases toward the periphery (Oliver & Webb, 2003). In younger, healthy individuals, the reflected waves travel back to the central arteries during diastole, enhancing coronary perfusion without increasing left ventricle afterload. Further along the vascular tree, the reflected waves occur earlier in the cardiac cycle due to differing elastic properties and, as a result, amplifying systolic pressure farther into the periphery. PP is amplified as well in the periphery with diastolic pressures progressively declining as the reflected waves occur earlier in the cardiac cycle (Oliver & Webb, 2003; Schiffrin, 2004).

As a result of increased arterial stiffness in the central arteries, incident waves from the aorta travel much faster and are reflected back at greater speeds arriving not in diastole but in systole (Oliver & Webb, 2003; Schiffrin, 2004). This further increases aortic systolic and left ventricle pressures, in turn, increasing ventricular workload leading to left ventricle hypertrophy (Oliver & Webb, 2003). Simultaneously, this lowers diastolic pressures which compromise coronary blood flow leading to ischemia and unstable capillary blood flow. PPs are increased and there is less amplification from

central to peripheral vessels with the waveforms becoming similar (Oliver & Webb, 2003; Schiffrin, 2004). Therefore, the velocity of the pressure waves (PWV) is indicative of arterial stiffness. The faster the velocity is of the pressure wave, the stiffer the artery. PWV can be determined non-invasively by dividing the distance between two reference points by the time delay of the recorded pressure wave at these two points (Oliver & Webb, 2003; Schiffrin, 2004). Typically, young (less than 24 years of age) central arteries will have a PWV value of 5 m/s (O'Rourke & Mancia, 1999).

#### *2.2.1.2 Arterial Compliance and Distensibility*

Arterial compliance and distensibility are both ways of describing the elastic properties of the arterial system. Distensibility, independent of artery size, is the ability of the vessels to become stretched and is defined as the relative change in area for a given pressure change:

$$\frac{((sCSA - dCSA) / dCSA)}{(P_s - P_d)}$$

Compliance alternatively is dependent on artery size and is defined as the absolute change in vessel area for a given change in pressure:

$$\frac{(sCSA - dCSA)}{(P_s - P_d)}$$

where sCSA and dCSA are systolic and diastolic cross-sectional areas of an artery and  $P_s$  and  $P_d$  are systolic and diastolic pressures, respectively (O'Rourke, Staessen, Vlachopoulos, Duprez, & Plante, 2002). Non-invasive Echo-Doppler ultrasound techniques are used to determine the above values. These viscoelastic properties can be used in order to determine the state and efficiency of the arterial system; the more

compliant and distensible an artery, the less stiff the arterial wall is and vice versa. A decrease in either compliance or distensibility have been recognized as important predictors of CVD and are associated with numerous CVD risk factors such as obesity (Tounian et al., 2001), hypertension (Urbina et al., 2011; Litwin et al., 2004; Gil, Sung, Shim & Hong, 2008), diabetes (Urbina et al., 2009), and age (van der Heijden-Spek et al., 2000; Gardner & Parker, 2010).

### *2.2.1.3 Intima-Media Thickness*

Another non-invasive marker of functional and structural arterial wall properties is IMT. IMT is a measure which represents the thickness of the intima and media layer of the arterial wall. It has become a method of choice for detecting subclinical diseases and their progression. A thicker intima-media has been considered one of the first signs of early atherosclerosis, induced by damaging factors such as high BP, lipids and increased body mass index (BMI) (Litwin et al., 2004). It has proven to be an independent risk factor for cardiac infarction and stroke, and is increased in patients alongside the number of cardiovascular risk factors attained (Bots, Hoes, Koudstaal, Hofman, & Grobbee, 1997). Therefore, IMT is widely used to evaluate the effects of risk factor modification on the progression of early arterial wall changes during intervention (Poredos, 2004). To assess IMT B-mode ultrasound is most commonly used on the left and/or right common carotid artery (CCA). The CCA is most suitable because of its superficial location, its larger size and limited movement (Poredos, 2004). Furthermore, the far wall of the carotid artery is better for identifying different arterial layer interfaces so is preferred over the near wall. Some investigators prefer to measure both the near and far wall enhancing precision; however, depending on the quality of the image, this may or may

not be possible (Poredos, 2004). B-mode analysis can be complicated because of the speckled pattern but by averaging the IMT along a continuous segment, it reduces the influence of speckling (Van Bortol et al., 2001).

### ***2.2.2 Echo-Doppler Ultrasound***

To assess and monitor arterial function and health, most methods are based on ultrasound techniques. The reasons for using ultrasound are mainly due to its non-invasive and non-traumatic nature, as well as for its extensive availability (Hoskins, Martin, & Thrush, 2010). Echo-Doppler ultrasound can be performed for purposes of measuring arterial stiffness and thickness; evaluating CVD risk in children and adults. Its use is limited to the larger more accessible arteries; therefore, in order to use Echo-Doppler ultrasound as a tool, one must first understand the concepts behind it and its limitations.

#### ***2.2.2.1 Principles***

Ultrasound consists of a high frequency sound wave which can be used to form images of internal body organs and structures. The frequency of an ultrasound is determined by its source and is defined as the number of wave lengths per second (hertz; Hz) and by the equation:

$$f = c/\lambda,$$

where  $f$  is frequency,  $c$  is the speed of sound, and  $\lambda$  is the wavelength. A frequency between 20 Hz - 20 kHz can be heard by the human ear; anything above 20 kHz cannot be heard and is therefore referred to as an ultrasound wave (Hoskins, Martin, & Thrush, 2010). In medicine, the range of frequencies used are between 2 MHz – 10 MHz and are

able to travel through bodily tissues propagating at the speed of sound ( $c$ ; for human tissues  $c = 1540\text{m/s}$ ) (Hoskins, Martin, & Thrush, 2010; Nichols & O'Rourke, 2005; White & Hollier, 2005). Attenuation in ultrasound describes the reduction in amplitude of the ultrasound wave as it travels through different tissues and/or scatters (Nichols & O'Rourke, 2005). Attenuation or reduced amplitude can affect the quality of the image and depends on the absorptive properties of the medium and the frequency of the wave. Therefore, higher frequencies (5 to 10 MHz) are used for the best resolution but waveforms at these frequencies increase attenuation and does not allow for deep penetration. However, lower frequencies (1.0 to 4 MHz) have longer wavelengths and are used for deep penetration (Nichols & O'Rourke, 2005). Thus, for superficial vessels such as the CCA, the frequencies usually range from 7.5-10 MHz (White & Hollier, 2005). Lastly, acoustic impedance represents the total resistance that the ultrasound is faced with while traveling through the different tissues. If there is a greater impedance as the waves travel through different tissues, then less waves are transmitted and more reflected-increasing attenuation. Correspondingly, higher frequencies also result in increased impedance (Hoskins, Martin, & Thrush, 2010).

#### *2.2.2.2 Transducers*

The key component to an ultrasound unit is the transducer. This device is what converts the electrically transmitted pulses into ultrasonic pulses and vice versa acting as both a transmitter and a receiver. The actual element that generates the sound and detects the signal is the piezoelectric plate inside the transducer. The material most used in medical imaging to make these plates is a synthetic ceramic lead zirconate titanate. Both sides of this plate are coated with conductive paint forming electrodes. Vibrating voltages

with positive and negative charges are applied to these electrodes, making the piezoelectric plate material expand and/or contract at a determined frequency and as a result transmit sound waves through the tissue. These waves are directed along a narrow beam, and its width and thickness being important determinants of acoustic noise in the image (Hoskins, Martin, & Thrush, 2010). A straight linear array transducer is designed for superficial imaging of central and peripheral arteries producing a larger, rectangular field of view. It is a high frequency probe, providing better resolution, but less penetration. Phased array transducers, in comparison, produce a pie-shaped image most beneficial for deep cardiac imaging (Nichols & O'Rourke, 2005).

### *2.2.2.3 Ultrasound Imaging Modes*

There are several display modes for ultrasound imaging: A-mode, B-mode and M-mode. A-mode (amplitude-modulated) is used to detect the distance of a single target (Nichols & O'Rourke, 2005). B-mode imaging represents brightness and records the reflected echo pulse as a dot creating a two-dimensional image. The brightness is related to the intensity/strength of the reflected signal after accounting for attenuation due to intervening tissue (Nichols & O'Rourke, 2005). B-mode machines now allow real-time display, producing approximately 30 images per second (Hoskins, Martin, & Thrush, 2010). M-mode is used to follow the motion of structures, represented by a frozen image at one point in time (Nichols & O'Rourke, 2005).

Doppler is another mode which typically measures velocity of blood flow using the frequency of sound reflected from moving red blood cells (Nichols & O'Rourke, 2005). The most commonly used modes of Doppler are pulsed (PW) and continuous

wave (CW). CW uses two separate transducers, one for transmitting and one for receiving continuous signals simultaneously. The Doppler signals are obtained from any reflectors in the area of where both beams cross paths, examining the maximum velocity along the entire scan (Nichols & O'Rourke, 2005). The closer the reflecting target, the higher the signal; the closer the target is to the centre of the beam, the stronger the signal. CW Doppler is relatively inexpensive, however, is not able to determine specific locations or be used to select a desired signal especially when structures, like vessels, can be so close to one another (Nichols & O'Rourke, 2005).

PW uses one transducer which transmits and receives short bursts of pulses at different times (Nichols & O'Rourke, 2005). Unlike CW Doppler, the PW Doppler method is accommodating. The length of the transmitted and received pulse determines the size of the sample volume. Its position along the sound beam is dependent on the time delay between transmission and reception (Nichols & O'Rourke, 2005). This control allows a PW Doppler to select well-defined areas of interest within a small and controllable sample volume, preventing interference by signals from adjacent vessels (Nichols & O'Rourke, 2005). Unfortunately, there is a limitation with the PW Doppler method where the maximum velocity is limited. If blood is travelling faster than half the pulse repetition rate (the number of pulses transmitted per second), ambiguity in the Doppler signal results (Nichols & O'Rourke, 2005). This ambiguity is also known as aliasing where the frequency of the Doppler signal is distorted. On the other hand, when the pulse repetition rate is too fast it can result in an overlap of transmitted echoes. To avoid this aliasing a signal must be sampled with a frequency, known as the Nyquist limit, at least twice the frequency of the signal itself (Nichols & O'Rourke, 2005).

#### *2.2.2.4 Limitations*

Since the methods of this thesis specifically involve imaging of the CCA, there are some limitations that should be mentioned concerning this technique. First, it is important to have an experienced operator in order to image the arteries accurately with adequate reproducibility (Mackenzie, Wilkinson, & Cockcroft, 2002). If the transducer is not held at the right angle to the artery of interest ( $90^\circ$ ), it may distort the image and become difficult to see the vessel in its entirety (Gill, 1985). It is also important that the arterial image run horizontally across the Doppler unit monitor and is in the focal zone for accurate analysis of arterial diameter changes during each cardiac cycle (Gill, 1985). Gain adjustments are also necessary for image quality and can help give clear identification of the intima-media. Lastly, it is important that the operator give attention to how much pressure is being applied onto the artery while imaging. If the hold-down pressure is too great, it may interfere with the pulsatility of the vessel inhibiting the measurement of true diameter changes. Echo-Doppler, when used correctly, can give information on arterial stiffness and thickness in children and adults, assessing and monitoring arterial function and health.

These non-invasive arterial health measures are what allow early assessment of cardiovascular health and provide a tool for monitoring and quantifying the effects of an intervention. However, before this is possible, it is necessary to understand the normal age- and sex- related differences within measures of arterial health. Only after this, can one properly assess the effects of an intervention and whether or not they produce differing results dependent on age and/or sex. However, research on children is not as extensive and conclusive as adults. Therefore, sex differences and the effect of aging will



briefly be discussed in adults first to show what is known in this area. Also, there is an extensive amount of evidence on psychosocial factors and their relationship to health in adults which is lacking in children. It is necessary to understand adult sex- and age-related changes relative to those in children in order to propose that similar relationships found in adults can be hypothesized in children.

### ***2.2.3 Arterial Health Measures in Adults***

Aging is a major determinant of arterial stiffness, altering the arterial wall as a result of tissue fatigue. In adults, it has been consistently found that compliance and distensibility both decrease with age and that arterial stiffness increases (Arnett, Evans, & Riley, 1994; Gardner & Parker, 2010; van der Heijden-Spek et al., 2000). Progressive degeneration of the arterial media affects the arrangement of elastic fibres causing fractures and fragmentation of the lamellae (layers of elastic tissue which separate the intima from the media). In parallel, there is an increase in collagen and calcium content accompanied by an accumulation of vascular smooth muscle cells in the walls of the artery. Also, as a result of elastic degeneration, there is arterial diameter enlargement (London & Guerin, 1999; van der Heijden-Spek et al., 2000; Hansen, Mangell, Sonesson, & Lanne, 1995). The consequence of the morphology of these elastic fibres is that arterial stiffness is enhanced. Age-related increases in stiffness amplify the pulse wave centrally (not peripherally), where PP of peripheral arteries and central arteries progressively become equal (Arnett, Evans, & Riley, 1994). Additionally, there is much evidence to support a reduction in endothelial function with age, which may be another determinant of arterial stiffening. Synthesis of NO has been reported to decline with increasing age because of a number of related mechanisms including, among others, a reduced

expression of NO synthase and an increase in reactive oxygen species (Muller-Delp, 2011). An increase in IMT can also be a result of normal aging in adults; as one gets older, the thickness of the arterial walls significantly increase. This is suggested to be a result of BP load and accumulation of smooth muscle cells with degeneration of elastic fibres (Ishizu et al., 2004).

In adults, evidence of this progressive increase in arterial stiffness and IMT with age is consistent throughout the literature; however, differences between the sexes are less clear. Elderly postmenopausal women seem to have an increased incidence of cerebrovascular and cardiac events compared to men of the same age (Berry et al., 2004; Ellekjaer, Holmen, Indredavik, & Terent, 1997). This may be due to the larger increase in incidence of isolated systolic hypertension found in postmenopausal women as a consequence of higher arterial stiffness (Berry et al., 2004). Berry and colleagues (2004) reported a lower compliance and distensibility in postmenopausal women compared to men of the same age, and an increase in AIX with sex being an independent determinant along with height and heart rate (HR). Premenopausal women have been shown to have higher compliance compared to age-matched men (Laogun & Gosling, 1982; Baskett, Lewis, Beasley & Gosling, 1990). This would suggest a postmenopausal hormonal effect, possibly the role of diminished estrogen. There have been many studies suggesting a cardiovascular protective effect of estrogen on the vascular system that may be exerted directly on the vessel wall. Administration of estrogen promotes vasodilation which may be due, in part, to an increased stimulation of NO production. With menopause, an altered amount of estrogen could diminish its protective effect, leading to an increase in arterial stiffness. However, the literature has been inconsistent where investigators have found

males to have increased arterial stiffness at ages 15 and 70 years compared to women of the same age (Hansen, Mangell, Sonesson, & Lanne, 1995), while others have found no sex differences (van der Heijden-Spek et al., 2000). In terms of sex differences in IMT, adult males have been found to have thicker arterial walls compared to age-matched women between 35-54 years (Juonala et al., 2008; Sass et al., 1998). It has been suggested that this difference may be attributed to the differences in cardiovascular risk factor levels and/ or body size (Juonala et al., 2008). To support this suggestion, a study done by Sass et al. (1998) found that arterial diameter was positively correlated with IMT- both larger in males aged 35-54 years. They also found these males to have significantly higher BMI and BP, both shown to be positively related to diameter and IMT (Sass et al., 1998). Again this difference may also be explained by the influence of hormones having a direct impact on the arterial wall (Sass et al., 1998). However, few studies take into account menopausal status and whether postmenopausal women are undergoing hormone replacement therapy. In fact, studies have shown that menopausal women undergoing hormone replacement therapy and/or temoxifen (estrogen- receptor modulator) therapy have lower IMTs compare to controls (Tremollieres et al., 2000; Simon et al., 2002).

The influence of sex hormones on arterial stiffness and thickness has mainly focused on estrogen. Estrogen has been shown to have a direct effect on arterial health with hormone receptors being found in the arterial wall (Fischer, Bashey, Rosenbaum & Lyttle, 1985). Estrogen has been shown to inhibit the increase in collagen when an atherogenic diet was introduced to female rabbits (Fischer, Bashey, Rosenbaum & Lyttle, 1985). Additionally, estrogen has been found to stimulate the release of NO as well as

being associated with lower levels of endothelin-1 (Harris & Matthews, 2004). It is also suggested that estrogen may influence the autonomic nervous system by up-regulating parasympathetic nervous system activity, while down-regulating sympathetic activity, although there are mixed results (Harris & Matthews, 2004). In addition, estrogen has been found to inhibit smooth muscle cell proliferation (Vargas, Wroblewska, Rego, Hatch, & Ramwell, 1993) and improve lipid profiles (Andersson et al., 1997). In comparison, increased levels of androgens which occur during male puberty have been associated with increased monocyte adhesion to endothelial cells; having a proatherogenic effect and promoting arterial stiffness (McCrohon, Jessup, Handelsman, & Celermajer, 1999). Investigators have also found an association between androgen deprivation and improved endothelial function in older men (McCrohon, Jessup, Handelsman, & Celermajer, 1999). A study done in female rabbits found the greatest degree of atherosclerosis in those treated with testosterone and progesterone, as well as increased collagen synthesis present in the aorta compared to controls (Fischer, Bashey, Rosenbaum & Lyttle, 1985). The increase in collagen was not directly linked to the increased degree of atherosclerosis but the two are hypothesized to be associated with one another (Fischer, Bashey, Rosenbaum & Lyttle, 1985). Therefore, overall the literature suggests that sex hormones play a significant role in arterial stiffness and thickness.

Sex hormones may also play a role in sex differences between children at different maturational levels, which will be further discussed in the next section. In addition to changes in stiffness and IMT with age and between sexes, the presence of cardiovascular risk factors such as hypertension, hypercholesterolemia, and obesity have

been found to negatively influence these arterial indices (Schiffrin, 2004; Arnett, Evans & Riley, 1994; Grobbee & Bots, 1994; Iannuzzi et al., 2004; Litwin et al., 2004; Vercoza, Baldisserotto, Abaete de los Santos, Eduardo Poli-de-Figueiredo, & Otavio d'Avila, 2009). In fact, arterial stiffness and thickness have been found to be indicative of atherosclerosis burden, strongly correlate with CVD risk factors (Bots, Dijk, Oren & Grobbee, 2002) and predict cardiovascular events in asymptomatic adults (Fathi et al., 2004). In particular, we and others have found arterial stiffness and carotid IMT to be associated with hypercholesterolemia (Riggio et al., 2010), diabetes (Christen et al., 2010), obesity (Banach et al., 2010), hypertension (Resnick et al., 1997) and lack of PA (Woo et al. 1994) in children, adolescents and adults. How these risk factors influence the development of arterial stiffness and IMT in children will be explored below in more detail as they are the focus of this thesis.

#### ***2.2.4 Arterial Health Measures in Children***

Based on the arterial health measures explained earlier, age- and sex-related changes in children are somewhat different than those found in adults. This does not change the role that risk factors have on children in terms of predicting CVD in adulthood, but would explain differing results of interventions implemented in children versus adults. Therefore, literature discussion regarding normal growth and maturation in children is necessary.

##### ***2.2.4.1 Growth and Maturation in Children***

Little information is known about the age-associated changes in arterial elastic properties of children. A review by Fernhall and Agiovlasitis (2008) stated that arterial

compliance decreases by 10-28% between 2 and 50 years of age, concomitant with a 9-30% increase in elastic modulus and stiffness index (Fernhall & Agiovlasitis, 2008). These percentage changes vary between studies with different methods and stiffness indices; however, all show that arterial stiffness does increase with age among children (Fernhall & Agiovlasitis, 2008; Jourdan et al., 2005; Lenard, Studinger, Mersich, Kocsis, & Kollai, 2004; Van Merode, Hick, Hoeks, & Reneman, 1989; Baskett, Lewis, Beasley & Gosling, 1990). However, some investigators have found an increase in compliance with age until adulthood, where after - there is a decline in compliance with age (Gardner & Parker, 2010; Senzaki et al., 2002). Whether it is that arterial stiffness increases with age or decreases, it is hypothesized that a change in stiffness is evident between age groups. Therefore, it is necessary to control for age when studying children of different ages. Nevertheless, although studies report contradictory findings, the majority of them used large age ranges not accounting for maturational or sex differences. Additionally, because compliance is determined by vessel size, normal developmental changes may influence measurement outcomes when using compliance to measure arterial stiffness. Senzaki et al. (2002) found that there was an increase in compliance in children aged 6 months to 20 years but when normalized for body surface area, a means to take into account arterial growth, they found a decrease in compliance with age (Senzaki et al., 2002). This finding suggests that an increase in compliance is primarily a function of increased arterial size that accompanies normal child development. As well, this observation coincides with the reduction in distensibility (independent of arterial size) and increase in the elastic modulus (a measure of wall stiffness) observed with childhood age (Senzaki et al., 2002).

However, after adolescence, there is little change in arterial structure and body size. Therefore the effects of aging are similar to adulthood where increased stiffness is related to decreased compliance and distensibility (Senzaki et al., 2002). A study done by van Merode and colleagues in 1989 was one of the first studies to look at distensibility and cross-sectional compliance of the CCA in males aged 4-19 years (Van Merode, Hick, Hoeks, & Reneman, 1989). The children were allotted into three age groups (i.e., Group I: 4-9 years; Group II: 10-14 years; Group III: 15-19 years). The authors demonstrated that SBP, lumen diameter, and PP were significantly higher among adolescents (Group III) compared to the younger age groups. They also found a lower distensibility in Group III, with no significant difference in compliance. This was also thought to be due to larger lumen diameters, again being that compliance is dependent on arterial size. This finding aligns with those of Senzaki et al. (2002) in that an increase in compliance is believed to prevent an increase in afterload (the stress developed in the wall of the ventricle during ejection), thereby maintaining normal ventricular performance during childhood. In essence, children's capacity to store blood (with each heart beat) is preserved with an increase in arterial diameter with age despite the increase in stiffness of the vessel wall. Showing significant changes in arterial stiffness up to adolescence further supports the hypothesis that the vascular system does mature by adolescence (Van Merode, Hick, Hoeks, & Reneman, 1989).

The age trend for IMT in children is also controversial. Some have found an increase with age while others have found little change (Bohm, Hartmann, Buck, & Oberhoffer, 2009; Ishizu et al., 2004; Sass et al., 1998; Vercoza, Baldisserotto, Abaete de los Santos, Eduardo Poli-de-Figueiredo, & Otavio d'Avila, 2009; Jourdan et al., 2005).

Different results could be due to the different methods used and whether or not investigators account for sex and maturational differences. On one hand, with growth, increases in lumen diameter and IMT have been found to be correlated with increased circulating blood volume (Ishizu et al., 2004). Based on the law of LaPlace, an increase in diameter and an increase in pressure are proposed to promote arterial wall thickening to maintain constant wall tension (Glagov, Zarins, & Giddens, 1988). In healthy children, this would suggest that the underlying mechanism responsible for increasing IMT may be the process of growth and not a pathological one as is true for adults. In addition, IMT has also been positively associated with height, being found as a strong determinant in a number of studies (Bohm, Hartmann, Buck, & Oberhoffer, 2009; Ishizu et al., 2004; Jourdan et al., 2005). On the other hand, some researchers do not see any age-associated changes until adulthood which is considered to be 18 years and above (Sass et al., 1998).

For both arterial elastic properties and IMT, sex differences in children have been much debated. Some investigators have shown no sex-related differences in arterial thickness and elasticity (Jourdan et al., 2005; Sass et al., 1998; Ishizu et al., 2004; Lenard, Studinger, Mersich, Kocsis, & Kollai, 2004; van der Heijden-Spek et al., 2000), while others have found different results (Bohm, Hartmann, Buck, & Oberhoffer, 2009; Vercoza, Baldisserotto, Abaete de los Santos, Eduardo Poli-de-Figueiredo, & Otavio d'Avila, 2009; Ahimastos, Formosa, Dart, & Kingwell, 2003). In studies where a sex difference is found in IMT, boys seem to have thicker walls compared to girls of the same age (Bohm, Hartmann, Buck, & Oberhoffer, 2009; Vercoza, Baldisserotto, Abaete de los Santos, Eduardo Poli-de-Figueiredo, & Otavio d'Avila, 2009). In terms of arterial stiffness, sex differences are less defined in the literature and appear to be mainly



influenced by pubertal status. A study done by Ahimastos & colleagues (2003) looked at arterial compliance and PWV in pre-pubescent and post-pubescent children to investigate sex differences and whether these differences are due to sex hormones. These researchers found that pre-pubescent girls had stiffer arteries compared to boys, whereas post-puberty this difference no longer existed. The existing sex differences had equalled out where post-pubertal girls had shown an increase in compliance and a decrease in PWV, which was opposite than that of boys where an increase in PWV in post-pubertal boys was observed (Ahimastos, Formosa, Dart, & Kingwell, 2003). These findings suggest that there are inherent differences between girls and boys and that this difference is modified by sex hormones introduced at puberty. Interestingly, there are similarities between this study and those comparing pre-and post-menopausal women (Westendorp et al., 1999), which could be the reason some studies do not find sex differences in adults until menopause (Laogun & Gosling, 1982) when levels of sex hormones become altered. However, it is important to note that this is just a hypothesis; intrinsic differences are unknown and there is still no conclusive evidence. It is still necessary to control for sex and maturation when looking at the effects of an intervention on children of different ages with the occurrence of possible intrinsic and/or maturational differences between boys and girls. If a child is exposed to one or more cardiovascular risk factors, the effect of sex on normal growth and development could be affected. It is therefore important now to discuss risk factors and the damaging effects they have on arterial health.

### **2.3 Arterial Health Risk Factors**

Traditional risk factors of CVD such as hypertension, elevated low-density lipoproteins (LDL), low high-density lipoproteins (HDL), diabetes and obesity have been

found to have a detrimental impact on arterial stiffness and IMT starting in childhood and tracking into adulthood (Juonala et al., 2005; Li, Chen, Srinivasan, & Berenson, 2004; Raitakari et al., 2003). For example, obesity is a growing health concern among not only adults, but children as well. As a result, there is an increased likelihood of childhood obesity carrying on into adulthood, laying down the “metabolic groundwork” for adult CVD (Srinivasan, Bao, Wattigney, Berenson, 1996). Among the middle-aged and elderly population, individuals who are obese have been shown to have an increased risk for stroke, CVD, and all-cause mortality (Wildman et al., 2005). However, adult CVD is not the only concern for obese children; the possibility of obesity-related atherogenesis beginning in childhood is another. In fact, studies have found that childhood obesity has adverse effects on arterial health. Body composition measures including waist circumference, percent body fat, and BMI have all been found to positively correlate with arterial stiffness and IMT (Tounian et al., 2001; Sakuragi et al., 2009; Vercoza, Baldisserotto, Abaete de los Santos, Eduardo Poli-de-Figueiredo, & Otavio d’Avila, 2009; Bohm, Hartmann, Buck, & Oberhoffer, 2009; Jourdan et al., 2005; Sass et al., 1998). In fact, studies have found that obese children have significantly lower arterial compliance and distensibility and higher arterial stiffness than age-matched, non-obese controls (Iannuzzi et al., 2004; Wildman, Mackey, Bostom, Thompson, & Sutton-Tyrrell, 2003). Similarly, research done in our lab has found that even after controlling for age, sex and maturation obese children have a lower distensibility compared to normal weight controls (Banach et al., 2010). In addition, several studies have found that obese children have thicker arterial walls than their non-obese counterparts (Iannuzzi et al., 2004; Vercoza, Baldisserotto, Abaete de los Santos, Eduardo Poli-de-Figueiredo, & Otavio

d'Avila, 2009; Stabouli, Kotsis, Karagianni, Zakopoulos, & Konstantopoulos, 2012), although others have found contrasting results (Tounian et al., 2001) where there was no difference in IMT in obese versus non-obese children. The inconsistencies reported in the literature may be due to the duration and degree of obesity as well as the coexistence of hypertension and other concomitant cardiovascular risk factors. Therefore, it has been suggested that there may be a cumulative effect of obesity with other associated risk factors such as high LDL levels and hypertension, which also promote arterial dysfunction (Vercoza, Baldisserotto, Abaete de los Santos, Eduardo Poli-de-Figueiredo, & Otavio d'Avila, 2009). It may be that the long term effect of obesity starting in early childhood leads to an increase in IMT during adolescence, as there is clear evidence that obesity does increase wall stress and stiffness (Iannuzzi et al., 2004; Wildman, Mackey, Bostom, Thompson, & Sutton-Tyrrell, 2003; Stabouli, Kotsis, Karagianni, Zakopoulos, & Konstantopoulos, 2012). For example, Stabouli and colleagues (2012) found that obesity was an independent prognostic factor for IMT in hypertensive children aged 10-19 years, proving obesity is a major determinant of arterial structure and quite possibly the early development of hypertension (Stabouli, Kotsis, Karagianni, Zakopoulos, & Konstantopoulos, 2012). Hence, not only is childhood obesity linked to adverse changes in arterial health, it can further contribute to the development of other cardiovascular risk factors such as hypertension and diabetes, which creates a vicious cycle that leads to further increases in arterial stiffness and IMT (Torrance, McGuire, Lewanczuk, & McGavock, 2007).

Hypertension is another major determinant of increased arterial stiffness. Structural changes in the aortic and carotid wall due to aging are strongly accelerated in

the presence of essential hypertension (London & Guerin, 1999). An increase in mechanical stress causes hypertrophy of large arteries as well as collagen accumulation resulting in increased arterial stiffness (London & Guerin, 1999). Several investigators have found that children with hypertension have increased CCA IMT as well as decreased distensibility and elasticity (Litwin et al., 2004; Riley et al., 1986; Sorof, Alexandrov, Cardwell & Portman, 2003; Gil, Sung, Shim & Hong, 2008). However, as seen in adults, these findings may not be consistent when it comes to different locations on the arterial tree, such as the radial artery compared to the common carotid (Laurent et al., 1994; Laurent, 1995). The CCA lumen has been found to be increased in children with hypertension as well, which can affect values of compliance preventing investigators from seeing a decreased compliance compared to normotensives (Litwin et al., 2004). Elastic modulus, another measure of arterial stiffness not related to arterial diameter but reflecting elastic properties is found to be increased in children with hypertension (Litwin et al., 2004; Riley et al., 1986). Increases in IMT have been found to correlate with increases in BP (Jourdan et al., 2005). As well, both BP and IMT have been found to be co-determinants of arterial stiffness in adolescents (Jourdan et al., 2005). Increased SBP and PP together with age are the major factors contributing to increases in IMT within hypertensive patients (Poredos, 2004). PP has been consistently found as a strong predictor of IMT in children (Litwin et al., 2004; Litwin et al., 2006). Even in normotensive children, a high PP can lead to repetitive wall stress and increased carotid IMT (Litwin et al., 2004).

Furthermore, PWV is positively related to BP in children (Schack-Nielson, Molgaard, Larsen, Martyn, & Michaelsen, 2005) and has been shown to be increased

even at the early stages of pre-hypertension suggesting that elevated BP in youth (pre-hypertensive) may still have negative effects on arterial health (Urbina, Khourey, McCoy, Daniels, Kimball, & Dolan, 2011). This observation also suggests that arterial stiffness may be a factor in the development of hypertension and not a resulting symptom (Liao et al., 1999). Dernellis and Panaretou (2005) produced findings that support the latter where aortic stiffness predicts future increases in BP of normotensive adults. Furthermore, one study found increased IMT in normotensive children who had parents with essential hypertension (Zizek & Poredos, 2002). This supports the idea that, along with the development of arterial stiffness, IMT may also be related to the development of hypertension and not a resulting symptom. Both hypotheses further support the importance of using such measures to prevent future cardiovascular complications.

Diabetes is also associated with arterial stiffening. Type 1 and type 2 diabetics have been found, even in young children, to have increased arterial stiffness compared to age- and sex-matched controls (Benetos et al., 2002). Even among non-diabetic subjects, individuals with higher blood glucose levels have been found to have stiffer arteries (Benetos et al., 2002). Furthermore, indices of insulin resistance are positively related to arterial stiffness and IMT (Tounian et al., 2001). Diabetics free of CVD have been shown to have IMT progression rates ten times that of non-diabetic controls with concentration of blood glucose being an independent predictor (Depairon et al., 2000). Furthermore, hyperinsulinemia and hyperglycemia increase the stimulation of the renin-angiotensin-aldosterone system promoting vascular hypertrophy (Nickenig, Roling, Strehlow, Schnabel, & Bohm, 1998) and hyperinsulinemia itself has direct proliferative effects (Cusi et al., 2000). Impaired glucose tolerance can lead to non-enzymatic glycation of

proteins with covalent cross-linking of collagen (AGEs), which alter mechanical and structural properties of the arterial wall promoting an increase in arterial stiffness (Zieman, Melenovsky, & Kass, 2005). Likewise, children with familial hypercholesterolemia have been shown to have increased arterial stiffness and IMT related to the level of LDLs (Sorensen et al., 1994; Wiegman et al., 2004). However, some studies have not found any relationship between stiffness and IMT and levels of LDLs but have with endothelial dysfunction in children with familial hypercholesterolemia. Endothelial dysfunction then may result in increased stiffness and thickness indirectly (Aggoun et al., 2000).

The presence of such risk factors speeds up the disease process and normal aging which may lead to increased CVD morbidity and mortality if not treated. In children, certain interventions could have a positive influence on reducing exposure of obesity, hypertension, diabetes and hyperlipidemia. As a result arterial health in children may be improved by altering arterial stiffness and thickness and in turn prevent the early development of CVD in adulthood. Therefore, the benefits of these interventions will be further discussed below and by doing so highlighting the importance of their implementation during developmental ages.

### **Chapter 3.0 Literature Review: Arterial Health Risk Factor Interventions**

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#### **3.1 Lifestyle Interventions**

Lifestyle interventions, such as aerobic exercise training, weight loss, and/or dietary modifications, have been successful in improving arterial health and treating stiffened arteries associated with morbidity in adults (Tanaka & Safar, 2005). Tanaka et

al. (2000) found that three months of regular aerobic exercise can restore some of the loss of central arterial compliance in sedentary adult men. This finding was independent of changes in body weight, adiposity, BP, plasma cholesterol, or resting HR, indicating a direct effect of exercise on compliance in healthy adults (Tanaka et al., 2000). Mechanisms behind this short term response to aerobic exercise are believed to be an increase in NO production resulting in vasodilation and counteraction of NO-inhibiting oxidants such as superoxide (Reed et al., 2005). Additionally, aerobic exercise may directly reduce sympathetic-adrenergic tone of the smooth muscle cells in the arterial wall (Tanaka et al., 2000; Wilkinson, Franklin, & Cockcroft, 2004). Certain dietary modifications such as salt restricted diets have also been found to have a direct effect on decreasing arterial stiffness (Tanaka & Safar, 2005). As well, fish oils (n-3 fatty acids) and antioxidant vitamins potentially have the ability to reduce arterial stiffness; however, more research is needed before recommending these for prevention or treatment (Tanaka & Safar, 2005).

The positive outcomes of such lifestyle interventions in adults have led to researchers investigating the possibility of such outcomes in children, as to further improve arterial health and reduce stiffening that tracks into adulthood. However, studies examining these relationships in healthy children are limited. Schack-Nielson et al. (2005) found that, in 10 year old children, low levels of PA were negatively associated with arterial stiffness; measured as PWV (Schack-Nielson, Molgaard, Larsen, Martyn, & Michaelsen, 2005). In contrast, Reed et al. (2005) did not find any relationship between arterial compliance and PA using a self-report questionnaire. They did however show a positive relationship between aerobic fitness and compliance in 9-11 year olds. Children

who were in the highest quartile for aerobic fitness had the highest compliance and correspondingly those in the lowest quartile had the lowest compliance (Reed et al., 2005).

More information is available on the effect of lifestyle interventions in children with co-morbidities, such as obesity. Woo et al. (2004) examined whether diet alone or diet with exercise could reverse arterial dysfunction among obese children (Woo et al., 2004). They found that both interventions improved arterial function. However, combined diet and exercise resulted in a larger beneficial arterial effect, additionally showing long-term improvements in endothelial-dependent dilation. Similar results were also shown in a study conducted by Meyer and colleagues (2006), where carotid IMT and flow-mediated dilation in obese children (11-16 years) significantly improved following a six month exercise training program (Meyer, Kundt, Lenschow, Schuff-Werner, & Kienast, 2006). The findings of these studies highlight the benefits of diet, PA and aerobic fitness as a means of protecting arterial function in children.

Despite the known benefits of diet and exercise on cardiovascular health in adults and children, adherence to exercise programs and changing from a sedentary to an active lifestyle can be a challenge, especially in children. The Canadian guidelines state children aged 5-17 years should participate in a minimum of 60 minutes of vigorous-moderate physical activity daily (Canadian Society for Exercise Physiology, 2012). Canadian Health Measures Survey data from 2007 to 2009 indicated that only 7% of this age group achieve this level of activity (Colley et al., 2011). Therefore, even with on-going messages focussing on proximal causes of disease and related risk factors (i.e. poor diet, lack of exercise, hypertension, diabetes and obesity), it has not been enough to fully



resolve the problem of poor arterial health in childhood. Proximal causes are those which directly cause disease and are thereby the nearest to disease on the causal chain. However, further up this causal chain, there are other factors which cumulate to explain the development of CVD risk factors, in addition to the lack of exercise and poor diet (Link & Phelan, 1995). These other factors, involved in developing arterial disease and risk factors may be dependent on one's living environment, life circumstances and/or behaviours, and may also explain, in part, why there are a high percentage of children not attaining the recommended amount of daily exercise. Therefore, it is important to understand the larger picture asking the questions, "why is there a lack of exercise and poor diet?" and "what is it about people's life circumstances and behaviour that shapes their exposure to such individually-based risk factors such as, hypertension, diabetes and obesity?" By asking these questions we open the mind up to other factors, also known as distal factors, which may appear further up the causal chain and cumulate with those more proximal fully explaining exposure to CVD risk factors and the evidence of arterial stiffness and thickness in children. Efforts to reduce risk by changing lifestyle behaviours may be hopelessly ineffective if there is no clear understanding of the process that leads to exposure. We must search for the factors that put people at risk of these risk factors.

According to Carruthers & Hood (2007) focusing on these proximal, direct causes of disease, unfortunately, has not resulted in healthier people or communities (Carruthers & Hood, 2007). The World Health Organization supports an updated definition of health with the inclusion of physical, mental and social well-being, not only the absence of disease (Breslow, 1972). This focus on positive health and well-being, rather than only the negative, has encouraged medical sociologists and social epidemiologists to study the

causal role of risk factors on disease which occur further up the causal chain of disease. These factors can be classified as distal causes and by examining these it permits a better understanding of the process of risk exposure (Link & Phelan, 1995). Psychosocial factors (i.e. positive and negative emotions and stress) are an example of such distal risk factors and have demonstrated an indirect role on disease development (Link & Phelan, 1995; Bruce et al., 2009; Everson-Rose & Lewis, 2005; Strike & Steptoe, 2004). More specifically psychosocial factors have been shown to be related to certain traditional CVD risk factors that have been mentioned previously, such as hypertension and diabetes (Carnethon, Kinder, Fair, Stafford & Fortmann, 2003; Bruce et al., 2009; Yan et al., 2003). However, these psychosocial factors tend to be overlooked and have received less attention as to their effects on children's arterial health.

### **3.2 Behavioural Interventions**

The idea that psychosocial factors are those that differentially expose people to more proximal, individual-based risk factors is based on literature in behavioural science. This literature has shown a significant association of psychosocial factors to different disease outcomes (Bruce et al., 2009; Everson-Rose & Lewis, 2005; Strike & Steptoe, 2004; Carnethon, Kinder, Fair, Stafford & Fortmann, 2003). For example, psychosocial interventions have shown to improve quality of life in patients with coronary artery disease (Schneiderman, Antoni, Saab, & Ironson, 2001), cancer (Schneiderman, Antoni, Saab, & Ironson, 2001), and kidney disease (Bruce et al., 2009), and seem to also influence biological processes thought to enhance disease progression (Bruce et al., 2009; Everson-Rose & Lewis, 2005).

Research to date on psychosocial risk factors has mostly looked at the effects of psychosocial interventions as a therapy or treatment strategy to increase longevity and slow progression of disease that has already developed in adults (Sullivan et al., 2009). Studies looking at how children may benefit earlier on to prevent such exposure are scarce. Bruce and colleagues (2009) discuss how psychosocial factors can adversely affect the nervous and vascular systems placing adults at a greater risk for kidney disease (Bruce et al., 2009). In fact, they present a model of the relationship between psychosocial factors and kidney disease. Figure 3.1 is adapted from their model to outline such a process between psychosocial factors and CVD, a disease that shares similar risk factors as kidney disease. This figure explores the need for psychosocial behavioural interventions to prevent and/or slow down the progression of vascular disease

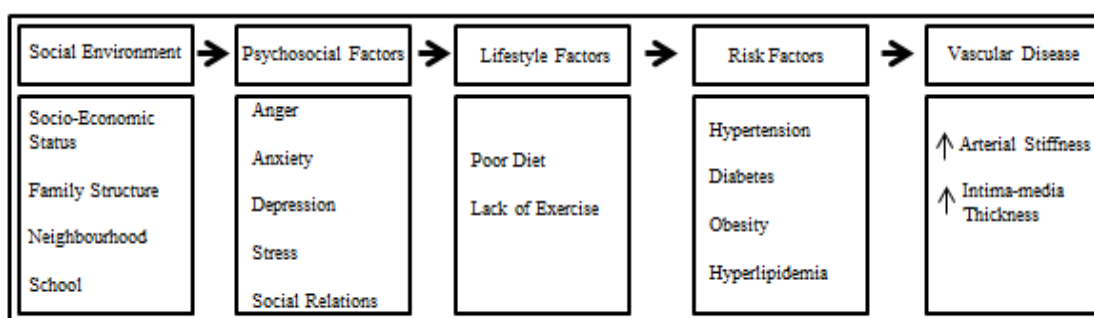


Figure 3.1. Pathway of associations between social environments, psychosocial factors, lifestyle factors, vascular disease risk factors, and vascular disease progression. Taken from Bruce et al. (2009).

development. The pathway also demonstrates that without understanding the psychosocial conditions that expose children to CVD risk factors, lifestyle interventions alone will fail more often than they should. This is because lifestyle interventions only fix those factors that are directly causing disease and related risk factors, they do not account for the persons social and psychological behaviours and life circumstances which have an indirect influence on arterial disease. If anything, changing lifestyle behaviours will only

temporarily fix the problem as underlining risk factors further up the causal chain should not be ignored.

Travelling further up the causal chain, social environmental factors such as socio-economic status and family structure seem to impact psychological functioning in adults as well as in children (Diez-Roux, Nieto, Tyroler, Crum & Szklo, 1995). Adapted by Bruce and colleagues (2009) Figure 3.1 suggests psychosocial factors are actually what bridge social environmental factors and disease outcome together (Bruce et al., 2009). While there is much research showing the relationship between health and socio-economic status (SES), an intervention at this level is impractical. This directs us to the even greater need to examine potential psychosocial behavioural interventions. Results of a psychosocial intervention increasing children's arterial health could provide the evidence of mechanisms linking psychosocial factors with CVD at the vascular level and would be important in making causal inferences. Researchers in therapeutic recreation have proposed the Leisure and Well-Being Model (LWM) which could provide an appropriate basis of the development of a psychosocial intervention to address cardiovascular health in children (Carruthers & Hood, 2007).

### ***3.2.1 Leisure and Well-Being Model***

The LVM is an effort to shift health research beyond direct risk factors to account for distal factors and the positive attributes of children that indirectly influence their overall physical, mental and social well-being. This model provides theoretical support for the role of psychosocial factors on health outcomes (Carruthers & Hood, 2007). By focusing only on direct/proximal risk factors, (e.g. lack of exercise, hypertension and

obesity) this will not entirely fix the problem of CVD risk factor exposure in children. Instead, the LWM indicates that it is necessary to directly assist in the development of environments and experiences that increase positive emotions, resources, and personal strengths that will influence more proximal risk factors to support the longer term goal of

improved health and well-being.

This supports the notion of examining distal factors such as psychosocial factors and how they may improve children's arterial health. Figure 3.2 is taken from a review of the LWM by Hood & Carruthers (2007) and better explains the fundamental basis of this model. To begin with, the long term goal of this model is to increase well-being. Having this endpoint in mind allows for the examination of other factors that may contribute to the attainment of this goal. Therefore, to assist one

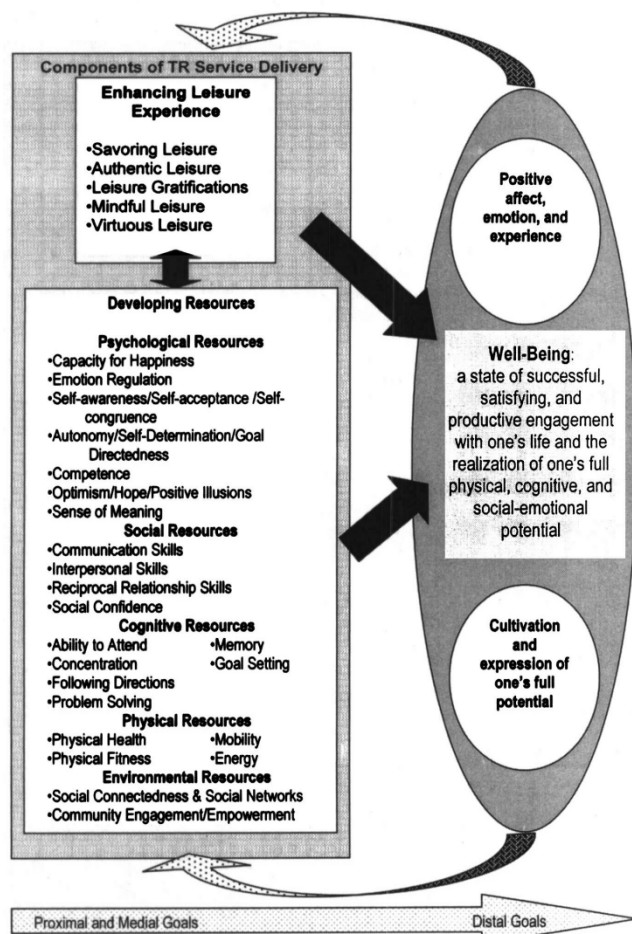


Figure 3.2. Components of the Leisure and Well-Being Model. Taken from Hood & Carruthers (2007).

along the pathway towards well-being, the two main components selected to be integrated into the LWM include: 1) experiencing positive affect, and emotion on a daily basis and 2) the cultivation and expression of one's full potential –including strengths,

capacities, and assets (Hood & Carruthers, 2007). Although these components are still considered long term goals, focusing on them will help to reach more proximal goals included in the model, maximizing the leisure experience and developing resources. These proximal goals are achieved sooner than those long term and on their own can elicit positive emotional experiences and capitalize on strengths. This interrelation, indicated by the arrows shown in Figure 3.2, triggers a positive upward spiral towards the enhancement of daily positive affect and experience and the realization of one's full potential; in the process this positive upward spiral contributes to improved health and well-being (Hood & Carruthers, 2007).

With the two main components of the model in mind (experiencing positive emotions on a daily basis, and cultivating one's full potential), one can now start to examine how the LWM can provide an appropriate theoretical base for a psychosocial intervention. Emphasis on both positive emotions and experiences and the realization of one's full potential can have a number of outcomes including health. Returning to the focus of this thesis- increasing children's arterial health- there may also be possible benefits of such psychosocial factors as positive emotion and knowing one's full potential on arterial health. To assess this, it is useful to examine the underlying components of the LWM with respect to child health, specifically child cardiovascular health.

### ***3.2.2 Components of the Leisure and Well-Being Model***

A psychosocial intervention based on the LWM focuses on four components which will all be discussed below and include: 1) positive emotions, 2) personal strengths, 3) coping, and 4) free-time vitality. The first component includes increasing

positive emotion, affect, and positive experiences on a daily basis (Carruthers & Hood, 2007). Positive emotion is viewed as an umbrella term used to address positive affect (the immediate physiological or emotional response) and various types of positive emotion, such as happiness and appreciation. The second component is the cultivation and expression of one's full potential, including strengths, capacities, and assets (Carruthers & Hood, 2007). For the most part, people want to do more than feel good; they want to develop their strengths and capacities in a personally meaningful way and feel that their lives have served some important purpose. The LWM refers to personal strengths as those found within the development of personal resources. A personal resource is those qualities and attributes that lie inside or outside the body drawn upon in times of need. The development of resources as sources of personal strengths allow for the creation of a positive and more enjoyable life. The third component integrated into a psychosocial intervention includes the development of coping strategies, used when confronted with life struggles. Based on the LWM, the development of personal resources act as a source of support when confronted with life stresses, and provides problem solving skills (Hood & Carruthers, 2007). It is important for a child to develop coping strategies as a way to deal with different stressful situations in their lives. Knowledge of one's personal strengths can act to help a person cope with life, allowing for an increase in one's protective factors such as, confidence and self-esteem when confronted with new challenges otherwise seen as stressful (Smith, 2006). Children who develop coping strategies are less likely to view a situation as stressful and are more likely to cope when stressful events do occur. The fourth and last component is increasing free-time vitality by maximizing leisure experiences. Leisure refers to those experiences that are pleasant

in expectation and involvement, intrinsically motivated and engaging. It includes play and recreation activities as well as other less structured and meaningful engagements (Hood & Carruthers, 2007). It is not only the participation in leisure, but also the quality of leisure which is an important aspect in the LWM. Choosing the right activities during free-time can increase a child's capacity to live, grow or develop. Moreover, if they are enjoying their activity, they are more likely to continue to do it.

As I discussed at length in the previous chapter, arterial health is defined by arterial stiffness and thickness. Further focus will be on the first two main components of the LWM, being positive emotions and personal strengths, with respect to their influence on child arterial health. Additionally, incorporating how maximizing leisure experience and developing coping strategies may interrelate, eliciting positive emotions and aiding in the development of new strengths. It will also be discussed on how leisure activities and coping may themselves influence arterial health, and how these interrelations may trigger an upward positive spiral towards the hypothesized increase in children's arterial health.

### *3.2.2.1 Positive Emotions*

Evidence of an association between positive emotions and health is limited. As a result, specific mechanisms that may link positive emotions to health have not been extensively studied. Studies have found a relationship between positive affect and better immune functioning (Salovey, Rothman, Detweiler & Steward, 2000), longevity (Danner, Snowdon & Friesen, 2001), successful aging (Rowe & Kahn, 1987) and, more specific to this thesis, cardiovascular function in adults (McCraty, Atkinson, Tiller, Rein & Watkins, 1995). McCraty and colleagues (1995) observed one possible mechanism which may



explain the protective effects on cardiovascular function. By facilitating positive emotional states, they found that subjects had an increase in baroreceptor activity- said to inhibit sympathetic nervous system activity and work as a controlling mechanism for BP (McCraty, Atkinson, Tiller, Rein & Watkins, 1995). Additionally, these authors previously found evidence of reductions in BP in hypertensive patients after they had learned to engage in positive emotional states. They suggested this finding was explained by the increase in baroreceptor activity (McCraty, Atkinson, Tiller, Rein & Watkins, 1995). When compared to the induction of negative emotions, two additional studies have found that activated positive emotions were associated with decreased BP (Knapp et al., 1992; Waldstein et al., 2000). As previously mentioned, hypertension and pre-hypertension have been found to be major determinants of arterial stiffness and IMT, thus the benefits of eliciting positive emotions (lowering BP) may indirectly restore stiffness in those with higher BP. In investigating the effects of positive emotions on arterial health in children, we must therefore make sure observed effects on arterial stiffness and thickness are not only specific for those classified with higher than normal BP.

Another mechanism, by which positive emotion might act as a tool in prevention of CVD indirectly, is by reducing levels of stress hormones. Overstimulation of the sympathetic nervous system results in an increase in HR and vasoconstriction which in turn increases vascular resistance and BP (McEwen & Seeman, 1999). Steptoe and colleagues (2005) found that middle-aged men and women in the higher happiness quintiles had lower levels of cortisol release during a working day and less of a fibrinogen stress response after performing a mental stress test. Both findings support the hypothesis that positive emotion is related to better cardiovascular health (Steptoe,

Wardle, & Marmot, 2005). The results of another study suggest that positive emotion may play a protective role in the development of hypertension, diabetes and respiratory tract infections, two of which are risk factors of arterial disease (Richman et al., 2005). These investigators found that higher levels of positive emotion were associated with a decreased risk of being diagnosed with hypertension two years later. Interestingly, after adjusting for negative emotions, the protective effects of positive emotion were unchanged indicating that the protective effects of positive emotion may be independent of negative emotional states (Richman et al., 2005). This suggests that what is responsible for the preventative effects on arterial health is not the lack of negative emotion but the facilitation and enhancement of positive emotion. This finding is in agreement with the work done by Bradburn and his associates whom also found that positive and negative affect were independent of each other and that both were separate predictors of self-reported happiness (Bradburn, 1969; Bradburn & Caplovitz, 1965). That would mean that feelings of unhappiness could be resolved without producing positive emotions and vice versa. Some health problems might result from the absence of positive emotion, whereas others may result from the presence of excessive negative affect.

An issue that arises with research examining the unique effects of positive emotion on health is the lack of consensus on what might be considered a positive emotion. Relative to negative emotions, positive emotions are fewer in number and are less differentiated (Fredrickson, 1998). The broaden-and-build model of emotion identifies four emotion families – joy, interest, contentment and love- and argues that within these emotion families, a variety of emotions may be characterized within a common theme (Fredrickson, 2001; 1998). Having such a broad definition is helpful

when such limited research is available. Fredrickson (1998) formulated this broaden-and-build model to better capture the unique effects of positive emotion. When one experiences a positive emotion, the urge to act out on these feelings triggers thought of possible courses of action. This thought process of nonspecific courses of action are referred to as momentary thought-action tendencies, and are the basis of Fredrickson's (1998) broaden-and-build model. The broaden-and-build theory states that certain positive emotions have the ability to broaden people's momentary thought-action tendency repertoires, widening the array of the thoughts and actions that come to mind. In contrast to negative emotion, which narrows thought-action repertoires and promote specific and immediate benefits, the broadened momentary thought-action repertoires elicited by positive emotions are beneficial in other ways. Specifically, broadened thinking serves to build personal resources, such as physical, social, and intellectual resources (Fredrickson, 2001; 1998). It is said that these personal resources developed during states of positive emotion outlast the transient emotional states that promote them and, if positive experiences are more frequent, personal resources will continue to build (Fredrickson, 2001; 1998). Personal resources act as sources of support and strength that lie inside or outside of the individual and are drawn upon in times of need (Hood & Carruthers, 2007). The unique effects of positive emotions – broadening thinking and building resources – overtime should accumulate, facilitating coping with life struggles and predicting future experiences of positive emotions. Notably, this elicits a positive upward spiral supporting the development of personal resources and their interrelation to positive emotions referring back to Figure 3.2 of the LWM.

Unfortunately, the majority of research on arterial health is not on how positive emotions promote health, but on how negative emotions promote CVD and its risk factors. In fact, numerous studies have found that negative emotions (depression, anger and anxiety) are associated with the morbidity and mortality of CVD (Strike & Steptoe, 2004; Everson-Rose & Lewis, 2005) and its risk factors such as diabetes (Carnethon, Kinder, Fair, Stafford & Fortmann, 2003) and hypertension (Yan et al., 2003; Davidson, Jonas, Dixon & Markovitz, 2000). It may then be assumed that those with a higher BP are subjected to a more negative emotional environment. Specifically, depression has been suggested to be a causative factor in the development of atherosclerosis and hypertension (Everson-Rose & Lewis, 2005; Strike & Steptoe, 2004; Yan et al., 2003). Therefore, benefits of lowering BP with the elicitation of positive emotions, previously described, may result in those with higher BP to experience a larger improvement in arterial health. This will be important to consider when looking at the effects of a psychosocial intervention on arterial health, investigating if children are affected differently, dependent on BP status. Both Rajagopalan et al. (2001) and Hemingway et al. (2003) have found that participants with depression had lower flow-mediated dilation than those without depression; having no other previous traditional cardiovascular risk factors. In addition, Rajagopalan et al. (2001) found that depression was also associated with increased levels of adhesion molecules (E-selectin, ICAM-1), suggesting a role in the development of inflammation leading to atherosclerosis and CVD (Rajagopalan et al., 2001). Hemingway and colleagues (2003) also examined the association between depression and other vascular measures of health, such as IMT and lumen diameters but did not find any differences between depressed and non-depressed individuals. These

authors suggest this finding could be a result of measurement error, and a small sample size. However, they also state no difference was seen in IMT because of the low overall risk of their sample reflected by a low mean IMT. This suggests that higher IMT found in depressed individuals may be due to the unhealthy lifestyles which follow as a result of depression (i.e. smoking and poor diet) (Hemingway et al., 2003).

To date, studies focussing on the effect of negative emotional states on direct measures of arterial health such as arterial stiffness and IMT are limited. Within this limited research, Paterniti and colleagues (2001) and Everson and his associates (1997) found that sustained anxiety and hopelessness in adults is a risk factor for the progression of atherosclerosis, observed by a greater increase in IMT over a 4 year period among those with higher levels of sustained anxiety and hopelessness (Paterniti, Zureik, Ducimetière, Touboul, Fève & Alperovitch, 2001; Everson, Kaplan, Goldberg, Salonen & Salonen, 1997). Yeragani et al. (2006) found that patients with anxiety disorders had higher measures of arterial stiffness compared to controls, measured via brachial-ankle PWV and arterial stiffness index. As well, two studies have shown a relation between anger and higher measures of stiffness (Anderson, Metter, Hougaku, & Najjar, 2006; Williams, Din-Dzietham, & Szklo, 2006). Only one study was found that examined the direct effects of a positive (laughter) psychosocial stimulus on direct measures of arterial health. This study, done by Vlachopoulos et al. (2009), found that after a 30 minute stimulus designed to provoke laughter, a decrease in PWV and wave reflections was observed at the end of the stimulus and for at least 30 minutes following. These observations were also found to be independent of changes in BP. Therefore, it is suggested that the reduction in arterial stiffness may mediate a reduction in CVD risk if

such stimuli were frequent; expected by those with increased positive emotions and experiences (Vlachopoulos et al., 2009).

The above findings support the link between positive emotions to arterial health. Whether positive emotions act independently or act to replace the harmful effects of negative emotions in children is unknown. However, both give reason to improve children's awareness, development and application of positive emotions. The literature above examined the effects of positive and negative emotions on arterial health in adults. If similar relationships are found in childhood the proposed psychosocial intervention could provide early prevention and decreased development of adverse effects on the arterial system. Work done by Midei and Matthews (2009) who looked at adolescents 14-16 years of age, found that those with higher levels of hostility and anxious attachment had greater values of PWV similar to previous findings in adults. Furthermore, they also found that those with higher levels of anger had greater central adiposity three years later, a marker which has been consistently found to be associated with an increased risk of arterial stiffness and CVD (Midei & Matthews, 2009). Unfortunately, research to date has only examined these negative emotions on children's arterial health and none were found investigating positive emotions.

One way to facilitate positive emotions in children is through maximizing the leisure experience. If the leisure activity is a positive experience, children may be more inclined to do the same activity again or to try new experiences which may further increase the level of challenge with a positive outlook. This continued positive outlook on life can have lasting impacts on health by engaging in other leisure activities and creating more opportunities to experience positive emotion. With the hypothesized preventative

effects of positive emotion on arterial health, maximizing leisure experience and increasing participation may indirectly, through increasing positive emotion and experiences on a daily basis, affect arterial health in children.

#### *3.2.2.2 Personal Strengths*

Similar to positive emotions, evidence of an association between cultivating personal strengths and health is also limited. There has been no research found on how personal strengths influence arterial health or prevents the development of CVD. However, not being aware of ones strengths affects the way a person views themselves on a daily basis, continually being reminded of failures or lack of success. Eventually this leads to diminishing self-confidence and self-worth and is a major source of stress (Pearlin, Libeberman, Menaghan & Mullan, 1981). The literature that is out there focuses more on these negative outcomes that may be a result from trying to deal with life events, working within one's limitations rather than one's strengths.

There is evidence that these negative outcomes, such as stress, have an influence on CVD development and more specifically on measures of arterial health. Stress is a normal human response that protects the body in the short run from threatening events and promotes adaptation, but in the long run can cause changes to the body that lead to disease. Higher levels of stress have been found to be a strong risk factor for the development of hypertension, atherosclerosis, and myocardial infarction (Das & O'Keefe, 2008). Continued high levels of stress can directly imbalance autonomic and hormonal homeostasis from chronic over activity of normal physiological responses (McEwen & Seeman, 1999). Repeated exposure to stress leads to increased wear and tear

of organs and/or tissues increasing the risk of developing disease. These imbalances can result in endothelial dysfunction, inflammation, and increased catecholamine release, further predisposing an individual to cardiovascular events (Das & O'Keefe, 2008). Previous studies have found that both catecholamine and endothelial dysfunction are regulators of arterial stiffness and wave reflection (Wilkinson et al., 2001; Wilkinson, Franklin, & Cockcroft, 2004). Therefore, this would explain the findings of Vlachopoulos et al. (2006) where they found a relationship between acute mental stress and increased arterial stiffness.

Stress-induced wear and tear over time, with exposure early in life, could reflect an acceleration of the aging process and also alter and remodel neuronal structures at sensitive developmental periods (Shonkoff, Boyce, & McEwen, 2009). Investigators have shown that there are maladaptive effects to the chronic release of stress hormones. These effects influence life-long alterations and lower thresholds in behavioural and physiological responses of children, such as anxiety and aggression (Shonkoff, Boyce, & McEwen, 2009). Therefore, reducing such sources of stress early on by helping children learn and develop their own personal strengths instead of reminding them of their weaknesses promises to be a powerful strategy for reducing, in part, the collective negative impact repeated stressors can have on arterial health's integrity in children. Moreover, developing and being aware of coping strategies would help children to reduce levels of perceived stress and prevent repeated exposure, indirectly improving arterial health. The development of both strengths and coping strategies allows children to be more confident in themselves and their abilities to succeed in all aspects of life with reduced exposure to daily stresses.



Leisure provides unique opportunities to undertake challenges, to develop strengths, try new things, build on personal resources, and act as a coping mechanism-providing an escape from negative emotion and on-going stresses (Hood & Carruthers, 2007). It should be the individual's decision on how they would like to spend their free-time, however, to maximize their experiences one would benefit more if the leisure activity chosen was in accordance with ones strengths, interests and goals (Hood & Carruthers, 2007). Among the different personal resources that are developed from participation in leisure, those of physical resources are included (refer back to Figure 3.2). Physical resources include physical health and fitness and are developed during leisure based PA. PA is linked to leisure by enjoyable experiences. In order for a child to initiate and sustain a physically active lifestyle, he or she must associate PA with enjoyment (Hood & Carruther, 2007). If a child is forced to participate and/or the activity is not within ones strengths but instead shows off their weaknesses, the likelihood of that child continuing that activity is low. Additionally, that child is less likely to explore other leisure activities with such a negative experience. With the hypothesized influence of coping and cultivating personal strengths on arterial health, maximizing leisure experience may indirectly-through a number of ways such as, increasing fitness and reducing exposure to daily stresses- affect arterial health in children.

### ***3.2.3 A Psychosocial Intervention***

Therefore, the intervention presented for this thesis is based on the LWM and consists of four units based on the components discussed above: 1) position emotion, 2) personal strength, 3) coping, and 4) free-time vitality. Each of the four units consists of their own learning activities that are hoped to improve children's awareness, skill

development and application in each unit. Full descriptions of these activities are provided in appendix E. The design of these activities were, in part, determined by youth and led by youth-for youth to ensure children's engagement. If activities were designed by adults they are less likely to relate to a younger age group (Caldwell, Darling, Payne, & Dowdy, 1999). As well, children may view adults as having control which would deter full participation. It is important that the children enjoy partaking in the intervention otherwise efforts of increasing positive emotions, developing strengths, coping strategies, and free-time vitality will have less of an impact on influencing arterial health. Additionally, to clarify that the hypothesized improvements on arterial health are not specific for children with higher BP, knowing the effects that positive emotions and coping with stresses can have on lowering BP, participants were stratified among BP groups (hypertensive, pre-hypertensive, and normotensive). The hypothesized improvements in children's arterial health would strongly support the influence of psychosocial factors on cardiovascular health. Additionally, examining the effects of a LWM-based psychosocial intervention on arterial health in children would provide new evidence of the benefits if such interventions were implemented, reducing the risk of disease progression in childhood which would otherwise further proceed into adulthood.

## **Chapter 4.0 Methods**

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### **4.1 Study Population**

The data used in this study was from a community-based study on the social determinants of BP in youth. The study involved children in grades five, six, and seven (aged 10 – 13 years) from one school board in Southern Ontario. There were a total of 50

elementary schools which provided consent and from those five that were stratified across SES (Rural, Urban Low SES & Urban High SES) were chosen to participate in the intervention, while another five schools were matched for comparison purposes. Being that SES may be a contributing factor to health and may lead to exposure of specific psychosocial factors, these pre-adjustments were made to prevent imbalances in the study design. The estimated population base was approximately 1044 subjects across the 10 schools identified. The study was approved by both Brock University and the school district research ethic review boards (refer to appendix A). Informed consent was obtained from the parent-guardian, and verbal assent was obtained from each youth to participate in the study (refer to appendix B).

Participation was voluntary but the sampling of participants to be included in this study occurred in two phases. First, of all students within the fifty schools whom provided consent, BP measurements were taken. Overall, 1913 students in grades 6, 7 & 8 had blood pressure measurements collected. This was done as phase one of the study in fall 2007/winter 2008 in order to generate a BP screening sample used to derive a BP percentiles table as reference for phase two of the study sample by their age, sex, and height (refer to appendix C). During phase two (spring 2010) a different sample of 647 children, within the 10 schools selected across grades 5, 6 & 7, volunteered and provided consent to undergo a school-based cardiovascular assessment which included a number of anthropometric and automated BP measures. Participants were then stratified by BP – those at or above the 95<sup>th</sup> percentile (HT; hypertensive), those at or above the 90<sup>th</sup> but less than the 95<sup>th</sup> percentile (PreHT; pre-hypertensive), and those below the 90<sup>th</sup> percentile (NT; normotensive) - based on the screening sample in phase one, and then randomly

selected from each of the BP groups to obtain a BP status stratified sample for additional testing. This randomization increased the likelihood that individuals would be comparable with respect to confounding variables that were not measured and could include: PA levels, diet, daily levels of positive and negative emotions, and stresses. In total, 111 (HT = 38, PreHT = 30, and NT = 43) children were included into the stratified sample and selected to participate in the additional cardiovascular physiology lab assessment that took place at each school. With the intention-to-treat, of the 111 participants that were selected, 60 (HT = 25, PreHT = 13, and NT = 22) underwent the intervention within the five participating schools while 51 (HT = 13, PreHT = 17, and NT = 21) from the five matched schools had no intervention implemented and were used as controls. There were no exclusion criteria for participation except refusal by the parent or student him/herself. Parents were allowed to withdraw their child from participating at any time.

#### **4.2 Measurements & Lab Protocol**

Prior to the school-based testing and intervention, parents provided consent for their children to participate in the study. All anthropometric, BP, and cardiovascular physiology measures were taken inside the participating elementary schools prior to and after the intervention in order to assess any changes. On the first visit children were removed from their classrooms in small groups and taken to a quiet room for BP and anthropometric testing. Participants rested in a seated position for 15 minutes, after which automated BP measurements were taken. Afterwards, height, sitting height, body mass and hip and waist circumference were collected. Skilled research assistants collected all data to ensure proper data collection. All assistants were required to attend training

sessions for each measure taken during the first visit. Upon each visit, research assistants were assigned to specific measures for that day to reduce variability. The cardiovascular physiology lab component was also carried out at the school approximately one to two weeks later on a sample of students selected at random and stratified across BP category. Specialized research assistants collected all data for the second visit to ensure proper cardiovascular data collection. If a participant was sick, a date was re-scheduled. Each measure is explained in more detail below (refer to appendix D for data collection recording sheets).

#### ***4.2.1 Cardiovascular Lab Measurement Protocol***

One to two weeks after the first testing visit, students who were randomly selected to participate in the cardiovascular lab component underwent a second day of testing done at their elementary school. Protocol was the same for each student and began first with a fifteen minute resting period in a supine position. Three manual BP measurements were taken one minute apart immediately before data collection. Data collection consisted of five minutes of beat-by-beat HR, BP, and PWV being recorded and monitored. Following beat-by-beat data collection, all CCA ultrasound images were taken. While images were being taken the participant was asked not to swallow or move and to stay quiet. At the end of testing three additional manual BP measurements were taken before allowing the participant to sit up. The same skilled research assistant was in charge of collecting all arterial images for all students to reduce variability in image quality. Another assistant was present to aid in the collection of all other measures. The total testing time for cardiovascular lab measurements was approximately 30-40 minutes per participant.

The four primary outcome/dependent measures used for analysis of arterial health were arterial compliance, distensibility, PWV and IMT as previously discussed. These outcome measures will be assessed to determine whether or not changes within one year are dependent on participation in the intervention.

#### ***4.2.2 Anthropometric and Maturational Measures***

Standing and sitting height were measured without footwear to the nearest 0.5 cm using a stadiometer (STAT 7X, Ellard Instrumentation Ltd., Monroe, Wash., USA). When determining sitting height, participants were seated with legs crossed, and leg length was calculated by subtracting sitting height from standing height. Body mass was measured to the nearest 0.1 kg using a digital scale (BWB-800S, Tanita Corporation, Tokyo, Japan). All measurements were taken three times and averaged for a final measurement. BMI was calculated as mass divided by height squared ( $\text{kg/m}^2$ ). The body composition measurements of waist and hip circumference were made by a female research assistant. Waist circumference was measured using a flexible-inelastic tape measure around the narrowest point of the waist to the nearest 0.1 cm. Waist circumference has been shown to provide an effective measure of central adiposity and fat distribution in children and adolescents as well as to be a strong indicator of other cardiovascular risk factors in adults (Daniels, Khoury, & Morrison, 2000; Dobbelsteyn, Joffres, MacLean, & Flowerdew, 2001; Taylor, Jones, Williams, & Goulding, 2000). Additionally, hip circumference was measured using a non-elastic tape measure around the greatest protrusion of the gluteal (buttock) muscles in order to calculate the waist-to-hip ratio (WHR).

Pubertal maturation was estimated from measurements of height, sitting height, leg length, body mass, and age (to the nearest 0.1 years), using the equation developed by Mirwald and colleagues (2002) that calculates years from peak height velocity (aPHV). Peak height velocity (PHV) represents the developmental period when growth of stature is greatest. The aPHV has been shown to be an accurate measure of somatic maturity offset, therefore representing the number of years before or after PHV occurs. In order to have a common classification between sexes, any negative offset prediction was classified as pre-PHV and any positive offset was classified as post-PHV (Mirwald, Baxter-Jones, Bailey, & Beunen, 2002). Additionally for the girls only, age of first menstruation was also recorded.

#### ***4.2.3 Blood Pressure and Heart Rate***

On the first visit to the schools, BP was recorded using an automated oscillometric BP device (BPM-300, VSM MedTech Devices Inc., Coquitlam, B.C., Canada). Measurements were taken consistently on the right arm with the cuff positioned at heart level. BP was taken after a 15 minute resting period with students in a sitting position with legs uncrossed, feet flat on the floor and hands supinated. Cuff size was based on arm circumference with at least 80% of the cuffs bladder encircling the arm and a cuff width that was at least 40% of mid-arm circumference (Pickering et al., 2005; Urbina et al., 2008). The inside of the cuff was marked with an index and range line. If the line was not within range, the cuff was replaced with an appropriate sized one. Six independent sequential measurements were then taken one minute apart. The first three measurements were used to familiarize the subjects with cuff pressurization and were

therefore discarded. The last three measurements were averaged to provide SBP and diastolic blood pressure (DBP) values.

During the cardiovascular lab component three measures of BP were taken one minute apart at the beginning of lab testing and again at the end of testing. Measurements were performed using a non-invasive standard sphygmomanometer placed on the right arm while participants rested in the supine position. The average of the three measurements taken at the beginning and end of lab testing were used to insure resting brachial artery BP was maintained throughout data collection. Continuous beat-by-beat non-invasive SBP and DBP was measured from the left middle finger using photoplethysmography (NexFin operations systems, Bmeye, Netherlands). Because BP taken at the finger slightly differs from that taken at the arm, the average of the last 3 manual BPs taken at the end of the beat-by-beat recording were used to adjust the beat-by-beat values collected simultaneously with photoplethysmography to ensure accuracy in children (Imholz, Settels, van der Meiracker, Wesseling, & Wieling, 1990). Additionally beat-by-beat pulse wave data was taken from the left middle toe (Pulse Oximeter, PB-11341031, Nellcor, Boulder, CO) and HR was recorded using a single lead electrocardiogram (ECG) waveform. Beat-by-beat data were collected using ADInstruments LabChart 7 Version 7.3.3 (ADInstruments, Colorado Springs, CO), and then transferred to Microsoft Excel (2007 version).

#### ***4.2.4 Arterial Stiffness: Compliance, Distensibility & Pulse Wave Velocity***

Central compliance and distensibility was measured at the CCA. The CCA was chosen for its large size and easy access. To measure CCA compliance and distensibility,



three non-invasive imaging sequences consisting of five beat-by-beat diameter changes in the right CCA were taken using Echo-Doppler ultrasound (Vivid I, GE Medical Systems, Netherlands). Images were taken approximately 3 cm proximal to the carotid artery bifurcation using an 8 MHz linear array transducer with diameter acquisition in B-mode. Once the carotid landmark was identified, continuous measures of the near and far walls were recorded. Diameters corresponding to systole and diastole were measured using manual callipers. The same investigator measured all diameter changes and was blind to the status of each subject. For each of the three CCA five beat image sequences the best two beats were chosen in terms of image quality and diameters were measured at three locations along the length of the artery in both diastole and systole. Therefore, for each subject 18 systolic and 18 diastolic diameter measures were averaged. Pulsatile cross sectional area (CSA;  $\pi r^2$ , where  $r = \text{diameter}/2$ ) and the corresponding pulse pressure was used in order to determine vessel compliance and distensibility using the standard equations:

$$\text{Compliance} = (\text{sCSA} - \text{dCSA}) / (P_s - P_d)$$

and

$$\text{Distensibility} = ((\text{sCSA} - \text{dCSA}) / \text{dCSA}) / (P_s - P_d)$$

where sCSA and dCSA are systolic and diastolic cross-sectional area and  $P_s$  and  $P_d$  are systolic and diastolic finger adjusted pressures, respectively. Although finger BP is known to be augmented compared to more central arterial BP (Oliver & Webb, 2003), it was the most accurate estimate of beat-by-beat PP. As well, intra-observer reliability of arterial diameters within our laboratory have shown very high intra-class correlation

coefficients for systolic diameter and diastolic diameter at 0.99, ( $p < 0.001$ ), and 0.96, ( $p < 0.001$ ) respectively (Phillips, 2009).

PWV was also measured as an indicator of arterial stiffness by taking the R-wave of the ECG as the starting point of our pulse wave and using the upstroke of the pulse wave taken from the left middle toe as our endpoint. The distance between the sternal notch and middle toe was used as the segment length. The PWV estimate for each subject was obtained by averaging a total of 10 beats using the following equation:

$$\text{PWV (cm/s)} = \text{segment length/transit time}$$

#### ***4.2.5 Intima Media Thickness***




IMT was measured using B-mode ultrasound on the far wall of the CCA as it provides more clarity (Wendelhag, Gustavsson, Suurkula, Berglund, & Wikstrand, 1991). To measure the thickness, the distance was measured from the luminal surface of the intima layer to the media-adventitia interface perpendicular to the longitudinal axis of the artery using an automated computerized edge-tracking method (EchoPac automated IMT package, GE Medical Systems, Netherlands). To ensure the lowest possible tension on the arterial wall, all measurements were completed at end-diastole. For each of the three five beat images of the CCA, IMT was measured on the two best quality beats at three locations (~ 3 cm in length) along the length of the artery, for a total of 18 IMT measures. The 18 measures were then averaged. All analysis of IMT was performed by the same investigator to ensure consistency. In order to test for repeatability, IMT was assessed previously in our lab. Intra-observer reliability showed very high intra-class correlation coefficients for IMT at 0.89 ( $p < 0.01$ ) (Phillips, 2009).

### **4.3 Intervention Implementation**

Students whom provided consent to participate in the psychosocial intervention were, at the time of preliminary testing, in grades five, six, and seven. This preliminary lab testing took place at the elementary schools from March 2010 to May 2010. Two months later, in July of 2010, fifty students finishing grade seven at the selected intervention schools were recommended by their teachers and invited to participate in a one week, overnight summer camp program held at Brock University. The goal of this summer camp was to design activities for a psychosocial intervention (designed by youth-for youth) which would be implemented in September of the 2010/2011 school year. Those who provided consent to participate in the summer camp program acted as peer health leaders during the intervention and were guided and assisted by qualified research assistant group leaders.

These grade seven students determined what content and methods they felt most effective at targeting the heart health of their peers, centred around areas previously identified based on the LWM- including: increasing positive emotion, personal strengths, coping strategies, and free-time vitality. Facilitators used materials gained from the elementary students to finalize a program suitable for five schools, and three grade levels. In order to assess the interventions' effectiveness, there needed to be some consistency between the schools. A lesson plan resource had been created outlining various activities to choose from for each unit (refer to appendix E). This way the peer health leaders had some choices in activities, but were all confined to a similar outline thereby remaining on a similar pathway as all other schools.

The intervention began in September of 2010 at the beginning of the new school year with students now in grades six, seven, and eight. An introductory video was made and presented to the students at each intervention school. This video provided the students with basic knowledge and understanding of the intervention and why it was being done. As well, it was meant to generate some enthusiasm and excitement for the months to come. Below is an outline of the structure for each of the four units discussed in the previous chapter, with the idea that each segment builds on top of each previous segment:

<b>Week</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5 / 0</b>
<b>Public Awareness</b>					
<b>Self-Awareness</b>					
<b>Skill Development</b>					
<b>Reflection/Application</b>					
<b>Research Team and Peer Leader Preparation</b>					<i>for next unit</i>

One month (approximately four weeks) was devoted to each unit of the intervention (increasing positive emotion, personal strengths, coping strategies, and free-time vitality), and each week of that month focused on a new segment (public awareness, self-awareness, skill development and reflection). Each weekly session was about 45-60 minutes long and usually occurred during the students lunch time so not to disrupt normal school curriculums. Additionally, research assistants also met once a week with the peer health leaders (selected grade seven and eights) to plan the sessions for the following

week. Research assistants also met with the research coordinator once a week to debrief, discuss issues and strategize for future sessions, making sure everyone was on the same level. Usually each school had two research assistants on each occasion and they remained constant over the year (some research assistants went to more than one school).

Post testing took place after the intervention was done six months later, one year after pre testing, in March 2011 to May 2011. As an incentive to participate in the intervention, every child eligible to participate was entered into a draw for a family package one-night stay at the Great Wolf Lodge in Niagara Falls, Ontario. Additional ballots were given at each phase of their participation in the study, increasing their chance of winning. This prize was awarded to one family from each of the 10 schools.

#### **4.4 Statistical Analysis**

All statistical analyses were carried out using SPSS software version 19.0 for Windows (SPSS Inc., Chicago, IL), and level of significance for all measures was set at  $p \leq 0.05$  (two-tailed). Participant characteristics in the intervention and comparison groups were examined using an independent-samples t-test and chi-square test for all variables as appropriate. Inclusion of participants within the intervention group was based on an intention-to-treat model of the school-based intervention. The effects of the LWM psychosocial intervention on all measures were determined using a two-way ANCOVA for repeated measures (time  $\times$  group) with sex and time-varying change scores (BMI, aPHV, BP, and HR only for PWV) included as covariates. The interaction of BP status with intervention groups was examined by a three-way ANCOVA for repeated measures (time  $\times$  group  $\times$  BP status) and included the same covariates as listed above for the two-

way repeated measures ANCOVA. However, before doing this a one-way ANOVA was performed to compare baseline values of all four of our primary outcome measures (compliance, distensibility, PWV, and IMT) between BP groups (HT, PreHT, and NT). Results are expressed as mean  $\pm$  standard deviation (SD).

## Chapter 5.0 Results

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### 5.1 Sample Characteristics

This study was part of a community-based, longitudinal study examining the effects of a psychosocial intervention on arterial health measures in children and adolescents. In total, from the 111 children whom were selected to participate in the additional cardiovascular assessment protocol, 82 (HT = 17, PreHT = 14, and NT = 51) children were included in the analysis. There was an attrition rate of 26% from year one to year two with 10 participants failing to complete the post-intervention measurements in 2011, and 19 whose beat-by-beat data were also incomplete. Those participants whom were lost to follow-up in year two were excluded from the study. Figure 5.1 displays a flow chart illustrating participants used for analysis.

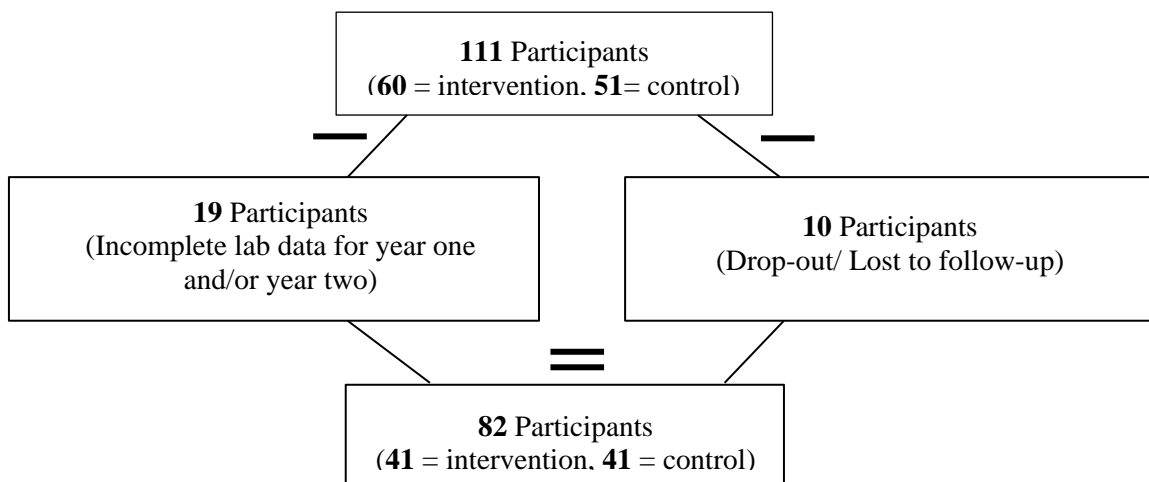


Figure 5.1. Flow chart illustrating the loss of participants.

An attrition analysis, using an independent-samples t-test and a chi-square test as appropriate, was performed to identify potential attrition bias comparing baseline values from those participants remaining to those excluded. Table 5.1 shows that automated SBP ( $t(40.89) = -3.095, p = 0.004$ ) and automated DBP ( $t(48.76) = -3.578, p = 0.001$ ) were significantly different between those who remained in the study to those excluded. It was also observed that BP groups were significantly different between those who remained to those excluded ( $\chi^2(2, N = 109) = 7.947, p = 0.019$ ). All other variables were not significantly different including age, sex, BMI, aPHV, height, weight, WHR, and HR. To further examine the difference between groups for SBP and DBP, a two-way ANOVA was performed to identify whether there were attrition differences in SBP and DBP across groups. The attrition x intervention interaction was significant for SBP only ( $F(1, 105) = 6.82, p = 0.010$ ). For the control group, the ones excluded from the study had higher SBP than those remaining in analysis while for the intervention group the SBP for those excluded was similar to those who remained.

With the intention-to-treat criteria, of the 82 participants that remained in the study 41 (HT = 12, PreHT = 5, and NT = 24) underwent the intervention within the five participating schools while 41 (HT = 5, PreHT = 9, and NT = 27) from five matched schools had no intervention implemented and were used as controls. First, an independent-samples t-test or chi-square test was performed as appropriate between the intervention and control groups to assess any baseline group differences. Table 5.2 shows the demographic and cardiovascular characteristics for intervention and control groups in year one. There was a significant difference between groups for sex ( $\chi^2(1, N = 82) = 5.125, p = 0.024$ ), aPHV ( $t(80) = 2.604, p = 0.011$ ), automated SBP ( $t(80) = -2.431, p =$

0.017), and automated DBP ( $t(80) = -2.045$ ,  $p = 0.044$ ). All other variables including age, BMI, height, weight, WHR, HR, and BP group were not significantly different between groups for year one.

Table 5.1. Baseline characteristics of those lost to follow-up, and those remaining in the study.

	Participants lost to follow-up	Remaining participants
N	29	82
Age (months)	142 ( $\pm 13$ )	144 ( $\pm 11$ )
Sex (%)		
Boys	44.8	61.0
Girls	55.2	39.0
BMI ( $\text{kg}/\text{m}^2$ )	23.1 ( $\pm 6.7$ )	21.1 ( $\pm 3.8$ )
aPHV (years)	-0.76 ( $\pm 1.23$ )	-1.2 ( $\pm 1.35$ )
Height (cm)	153.7 ( $\pm 10.4$ )	151.0 ( $\pm 9.9$ )
Weight (kg)	55.4 ( $\pm 19.7$ )	48.5 ( $\pm 11.2$ )
WHR	0.87 ( $\pm 0.08$ )	0.85 ( $\pm 0.07$ )
HR (beats/min)	86 ( $\pm 16$ )	83 ( $\pm 12$ )
SBP (mmHg)	103.2 ( $\pm 11.3$ )*	96.0 ( $\pm 8.9$ )
DBP (mmHg)	68.3 ( $\pm 10.1$ )*	60.50 ( $\pm 10.0$ )
BP Groups (%)		
NT	44.4*	62.2
PreHT	7.4*	17.1
HT	48.1*	20.7

Values are means ( $\pm$ SD), independent-samples t-test.

Note: \* Significant difference between groups ( $p < 0.05$ ). N = sample size, BMI = body mass index, aPHV = years from peak height velocity, WHR = waist-to-hip ratio, HR = heart rate, SBP = automated systolic blood pressure, and DBP = automated diastolic blood pressure, NT = normotensive, PreHT = pre-hypertensive, and HT = hypertensive.



Table 5.2. Demographic and cardiovascular measures in year one for intervention and control groups.

	Intervention	Control
N	41	41
Age (months)	142 ( $\pm 12$ )	145 ( $\pm 11$ )
Sex (%)		
Boys	73.2*	48.8
Girls	26.8*	51.2
BMI ( $\text{kg/m}^2$ )	21.0 ( $\pm 3.8$ )	21.2 ( $\pm 3.8$ )
aPHV (years)	-1.54 ( $\pm 1.39$ )*	-0.79 ( $\pm 1.22$ )
Height (cm)	149.7 ( $\pm 9.2$ )	152.4 ( $\pm 10.5$ )
Weight (kg)	47.5 ( $\pm 10.7$ )	49.5 ( $\pm 11.8$ )
WHR	0.85 ( $\pm 0.06$ )	0.85 ( $\pm 0.07$ )
HR (beats/min)	83 ( $\pm 14$ )	84 ( $\pm 10$ )
SBP (mmHg)	98.4 ( $\pm 8.1$ )*	93.7 ( $\pm 8.3$ )
DBP (mmHg)	62.7 ( $\pm 10.9$ )*	58.3 ( $\pm 8.6$ )
BP groups (%)		
NT	58.5	65.9
PreHT	12.2	22.0
HT	29.3	12.2

Values are means ( $\pm$ SD), independent-samples t-test & chi-square test for sex and BP groups.

Note: \* Significant difference between groups ( $p < 0.05$ ). N = sample size, BMI = body mass index, aPHV = years from peak height velocity, WHR = waist-to-hip ratio, HR = heart rate, SBP = automated systolic blood pressure, DBP = automated diastolic blood pressure, NT = normotensive, PreHT = pre-hypertensive, and HT = hypertensive.

## 5.2 Arterial Health Measurements

The mean values for all arterial health measurements are shown in Table 5.3 for the intervention and control group in year one and in year two. Sample sizes for each variable are the result of listwise deletion in cases where technical issues resulted in unsatisfactory arterial images, beat-by-beat BP, and beat-by-beat pulse wave data values required for the calculation of compliance, distensibility, IMT and PWV were inadequate, as well as missing covariate measurements.

Before assessing if there was an intervention effect after a one year time interval, an independent-samples t-test on the arterial health measures was done to assess if there were any baseline differences between the control and intervention groups. No significant differences were found. Next, a two-way ANCOVA for repeated measures analysis was done to measure if there was an intervention effect present. Results showed no significant time x group interactions for compliance, distensibility, PWV, and/or IMT. However, automated SBP did have a significant time x group interaction over time ( $F(1, 73) = 4.085, p = 0.047$ ). An illustration of this interaction is shown in Figure 5.2 and covariates included:  $\Delta$  aPHV,  $\Delta$  BMI, sex,  $\Delta$  automated SBP and  $\Delta$  automated DBP (excluding for SBP and DBP outcome variables), and HR only for PWV. aPHV is dependent on age for its calculation so age was not used as a covariate.

Table 5.3. Arterial health measurements between groups in year one and year two.

	Intervention		Control	
	Year One	Year Two	Year One	Year Two
Distensibility ( $\text{mmHg}^{-1} \times 10^{-2}$ )	0.96	0.82	1.01	0.87
(N = 75)	( $\pm 0.39$ )	( $\pm 0.27$ )	( $\pm 0.28$ )	( $\pm 0.23$ )
Compliance ( $\text{mm}^2/\text{mmHg}$ )	0.19	0.16	0.18	0.16
(N = 75)	( $\pm 0.08$ )	( $\pm 0.05$ )	( $\pm 0.05$ )	( $\pm 0.06$ )
PWV (cm/second)	371.1	371.8	371.4	372.4
(N = 72)	( $\pm 27.1$ )	( $\pm 27.8$ )	( $\pm 24.9$ )	( $\pm 25.6$ )
IMT (mm)	0.47	0.46	0.46	0.44
(N = 69)	( $\pm 0.06$ )	( $\pm 0.05$ )	( $\pm 0.05$ )	( $\pm 0.04$ )
SBP (mmHg) *	98.5	96.4	94.0	96.6
(N = 78)	( $\pm 9.0$ )	( $\pm 7.6$ )	( $\pm 8.4$ )	( $\pm 6.9$ )
DBP (mmHg)	63.5	60.4	58.5	59.1
(N = 78)	( $\pm 10.6$ )	( $\pm 7.3$ )	( $\pm 8.5$ )	( $\pm 6.1$ )

Values are means ( $\pm$ SD). Two-way ANOVA for repeated measures.

Note: \* Significant time by group interaction ( $p < 0.05$ ). PWV = pulse wave velocity, IMT = intima-media thickness, SBP = automated systolic blood pressure, and DBP = automated diastolic blood pressure. Controlling variables for:  $\Delta$  aPHV,  $\Delta$  BMI, sex,  $\Delta$  automated SBP and  $\Delta$  automated DBP (excluding for SBP and DBP outcome variables), and HR for PWV.

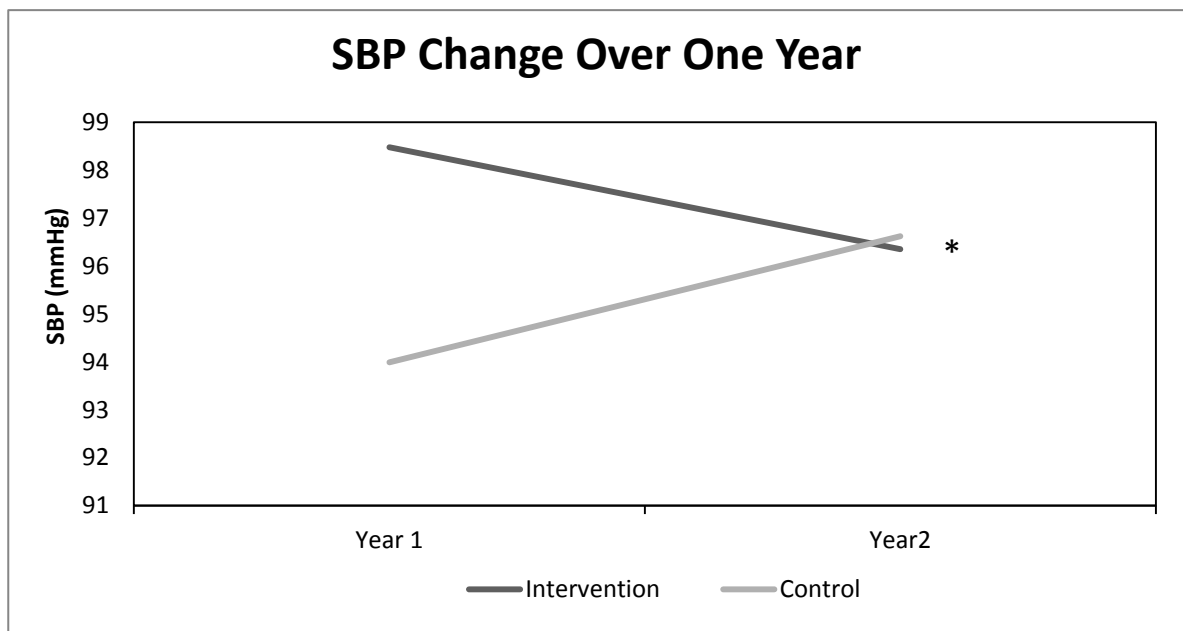


Figure 5.2. Change in automated SBP over a one year time interval between groups.

Note: \* Significant time by group interaction ( $p < 0.05$ ). SBP = systolic blood pressure.

Lastly, the 3-way interaction of BP status with intervention group over time was assessed in order to determine if those in the three different BP groups (HT, PreHT, and NT) were affected by the intervention differently. Table 5.4 shows the descriptive statistics of a one-way ANOVA which found baseline values of IMT ( $F(2, 97) = 3.69, p = 0.028$ ), automated SBP ( $F(2, 106) = 44.37, p = 0.0001$ ), and automated DBP ( $F(2, 106) = 32.63, p = 0.0001$ ) to be significantly different between BP groups. However, results of the three-way ANCOVA for repeated measures (time  $\times$  group  $\times$  BP status) showed no significant interactions in any of the outcome variables. Thus, the significant automated SBP time  $\times$  group interaction was not different between BP groups. Hence, if the intervention and control groups were each divided into their three BP groups, the results would look similar to those in Figure 5.2.

Table 5.4. Arterial health measurements between BP groups for year one.

	NT	PreHT	HT
N	51	14	17
Distensibility (mmHg <sup>-1</sup> $\times 10^{-2}$ )	1.03 ( $\pm 0.34$ )	1.01 ( $\pm 0.43$ )	0.91 ( $\pm 0.23$ )
Compliance (mm <sup>2</sup> /mmHg)	0.20 ( $\pm 0.07$ )	0.20 ( $\pm 0.08$ )	0.17 ( $\pm 0.05$ )
PWV (cm/second)	369.3 ( $\pm 26.9$ )	377.9 ( $\pm 23.3$ )	373.7 ( $\pm 26.6$ )
IMT (mm)	0.47 ( $\pm 0.04$ )	0.44 ( $\pm 0.04$ )	0.50 ( $\pm 0.07$ )**
SBP (mmHg) *	91.0 ( $\pm 6.1$ )	102.7 ( $\pm 4.3$ )	105.8 ( $\pm 7.1$ )
DBP (mmHg) *	56.4 ( $\pm 6.8$ )	65.3 ( $\pm 7.6$ )	68.9 ( $\pm 13.1$ )

Values are means ( $\pm$ SD). One-way ANOVA.

Note: \* Significant group effect ( $p < 0.05$ ). \*\* Significant difference between PreHT and HT groups ( $p < 0.05$ ). NT = normtensive, PreHT = pre-hypertensive, HT = hypertensive, N = sample size, PWV = pulse wave velocity, IMT = intima-media thickness, SBP = automated systolic blood pressure, and DBP = automated diastolic blood pressure.

## **Chapter 6.0 Discussion**

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### **6.1 Summary**

This study evaluated the effects of the LWM psychosocial intervention on arterial health, defined by the measure of arterial stiffness and thickness, in children and adolescents aged 10 -13 years. Measures of arterial stiffness and thickness included CCA compliance, distensibility and IMT using Echo-Doppler Ultrasound, and systemic PWV calculated from beat-by-beat pulse wave data. The LWM psychosocial intervention was to provide children with the awareness, skill development, and application of positive emotion, personal strengths, coping, and free-time vitality. We hypothesized that children who were exposed to the psychosocial intervention would have increased arterial health compared to those children who were not exposed to the intervention. To the author's knowledge, this study is the first study of its kind examining the effects of the LWM psychosocial intervention on arterial health in children. The principle findings of this study indicated that there was no increase in arterial health for children whom were exposed to the intervention compared to controls, with CCA compliance, distensibility, IMT and systemic PWV not statistically different between groups over one year. However, interestingly a significant difference between groups after one year (time x group interaction) was evident in SBP where it decreased in children exposed to the intervention and increased in those of the control group.

### **6.2 Arterial Health Measures**

Arterial stiffness and thickness are both very powerful predictors of CVD development. In children especially, these measures are useful because of their non-

invasive nature measuring arterial function and structure during developmental stages. Stiffness is one of the earliest detectable manifestations of adverse structural and functional changes within the arterial wall (Arnett, Evans, & Riley, 1994; Blacher et al., 1999). It has also been shown to be a modifiable measure sensitive to lifestyle and psychosocial factors previously described (Tanaka & Safar, 2005; Midei & Matthews, 2009; Vlachopoulos et al., 2006; Anderson, Metter, Hougaku, & Najjar, 2006; Williams, Din-Dzietham, & Szklo, 2006). Measures of stiffness done in this study included compliance, distensibility and PWV.

Compliance and distensibility both measure the elastic properties of the arterial wall and determine the state and efficiency of the arterial system. However, compliance is dependent on artery size and thus when measuring children undergoing normal developmental changes that include increases in arterial growth and lumen diameters, it must be interpreted with caution (Van Merode, Hick, Hoeks, & Reneman, 1989). Therefore, distensibility may be a more appropriate measure when looking at children. Even so, there was no difference between the intervention and control group over time with either measure of elasticity.

PWV is an indicator of stiffness measuring the velocity of the pressure waves emitted as the left ventricle ejects blood into the arterial system (Schiffrin, 2004). The way in which PWV was measured in this study, taken from the peak of the R-wave to the foot of the toe pulse wave, incorporates both systemic and central arterial stiffness into the measure. There are other methods of determining PWV that can independently assess central arterial stiffness however these measures are investigator dependent and time consuming as pulse waves must be collected simultaneously from both the CCA and

femoral artery using pressure sensitive probes. As well due to probe placement in the groin area, taking pulse wave measures at the femoral artery is very uncomfortable for children. Therefore, when dealing with children and a community-based study design where all testing was done at elementary schools during class time, our method was appropriate. In fact, our measure of PWV is a strength as it does incorporate the entire vascular system. Nevertheless, the one year LWM intervention did not affect PWV.

IMT represents the thickness of the intima and media layers of the arterial wall. It has proven to be a risk factor for cardiac infarction and stroke that can be modified by lifestyle factors and presumably psychosocial factors such as anxiety and hopelessness (Bots, Hoes, Koudstaal, Hofman, & Grobbee, 1997; Paterniti, Zureik, Ducimetière, Touboul, Fève & Alperovitch, 2001; Everson, Kaplan, Goldberg, Salonen & Salonen, 1997). However, most of this research has been in adults and no psychosocial intervention effect on CCA IMT was found in this study looking at children. Therefore, all four measures of arterial health in this study were not improved after exposure to a psychosocial intervention. To the author's knowledge no other studies have looked at changes in early arterial health indicators in response to a LWM psychosocial intervention in children. However, there have been many studies suggesting the potential preventative effects. The possible reasons for these findings, focusing back to the literature on psychosocial factors and arterial health, are discussed below.

### ***6.2.1 Arterial Health and Psychosocial Factors***

The literature is scarce regarding children on the subject of psychosocial factors and arterial health. Research has not specifically investigated the preventative effects that

a psychosocial intervention could have on measures of arterial stiffness and thickness during childhood and whether these benefits continue into adulthood. However, the amount of research relating psychosocial factors to all different areas of health- including cardiovascular disease, kidney disease and cancer- has led to a number of new questions and possibilities for shifting health research into taking a more serious look at distal causes of disease. The majority of research finding a relation between psychosocial factors and arterial health has been in adults, with a few studies done in children showing similar results. Midei and Matthews (2009) found that adolescents 14-16 years, whose levels of hostility and anxious attachment were higher, concurrently had greater values of PWV. Additionally, Shonkoff and colleagues (2009) found that there are maladaptive effects to the chronic release of stress hormones, such as lowering thresholds in behavioural and physiological responses of children, especially at sensitive developmental periods. This study, to the best knowledge of the author, is the first longitudinal study looking at the proposed preventative effects of a psychosocial intervention. The majority of researchers have published case-control studies looking at correlations between different arterial health measures such as PWV, endothelial function, IMT, and stiffness index, with specific psychosocial factors such as negative emotional states and stress (Stephoe, Wardle, & Marmot, 2005; Rajagopalan et al., 2001; Hemingway et al., 2003; Anderson, Metter, Hougaku, & Najjar, 2006; Williams, Dzitham, & Szklo, 2006). The LWM acts as an appropriate basis for the development of a psychosocial intervention in children encompassing positive emotions, personal strengths, coping, and free-time vitality in hopes to facilitate positive well-being, decrease negative emotion, and stress. As a result it was hypothesized that an increase in



arterial health would occur following a LWM intervention. Unfortunately, the results did not show a LWM psychosocial intervention effect on any of the arterial health measures. This does not mean that the relation between psychosocial factors and arterial health does not exist but, for a one year longitudinal study, it may not be enough time to see the potential changes.

Normal developmental changes in children at the arterial level are small especially in IMT. Cross-sectional studies looking at age trends for IMT that have found significant changes are over 2 - 4 years (Bohm, Hartmann, Buck, & Oberhoffer, 2009; Paterniti Paterniti, Zureik, Ducimetière, Touboul, Fève & Alperovitch, 2001). Bohm and colleagues (2009) only found a mean increase of 0.02 mm in two years. As well, when looking at normal and abnormal (diabetic and hypercholesterolemic) groups of children the mean differences are also very small (Ishizu et al., 2004). Being a longitudinal study, we were able to track the same children as they grow to see if these changes differed depending on exposure to the LWM psychosocial intervention. Following the participants for only one year may be a possible explanation as to why no effect was found. A longitudinal study looking at IMT changes between adults with sustained anxiety and adults without anxiety had followed subjects for 4 years and still only saw small changes between the two groups (approx.  $0.04 \pm 0.11$  mm) (Paterniti Paterniti, Zureik, Ducimetière, Touboul, Fève & Alperovitch, 2001).

Arterial stiffness, measured in this study by compliance, distensibility and PWV, seem to have notable changes throughout childhood. Compliance and distensibility are believed to decrease and PWV is believed to increase with age (Fernhall & Agiovlasitis, 2008; Jourdan et al., 2005; Lenard, Studinger, Mersich, Kocsis, & Kollai, 2004; Van

Merode, Hick, Hoeks, & Reneman, 1989; Baskett, Lewis, Beasley & Gosling, 1990). In this study, we were able to see a slight trending decrease in compliance and distensibility for both the control and intervention group shown in Table 5.3. However, there was no significant difference in the decrease across groups. Again, a one year intervention may not be enough time to see a significant psychosocial intervention effect on normal maturational changes. Moreover, as the participants were healthy children from a community sample, any differences may take longer to manifest.

Exercise intervention programs have been shown to have beneficial effects on reversing arterial dysfunction and improving IMT and arterial elasticity within 3-6 months (Tanaka et al., 2000; Meyer, Kundt, Lenschow, Schuff-Werner, & Kienast, 2006; Woo et al., 2004). However, many of these studies are in adults and those in children have focused on individuals whom have co-morbidities, such as obesity. Therefore, they are at a higher risk than those in this study whom are overall healthy children. Also, exercising techniques trigger a more direct and immediate response than psychosocial factors. The LWM psychosocial intervention was intended to increase arterial health, but is considered to be a more distal factor occurring further up the causal chain. Therefore, a LWM intervention may not have immediate effects intrinsically throughout the arterial system, but with a longer exposure beneficial changes may be evident. This type of intervention is an educational tool, teaching children ways to deal with stress, keep a positive attitude and capitalize on strengths in all aspects of life throughout childhood and into adulthood. It is intended to prevent disease by decreasing risk exposure and therefore cannot be compared to exercise interventions which mostly seek to reverse damage that is already present, failing to look at underlying distal factors.

### **6.3 BP and Psychosocial Factors**

In this study, although no LWM psychosocial intervention effect was found directly on arterial health measures, an effect was found on SBP. SBP is considered to be an important predictor of arterial stiffness and thickness and therefore plays a role in the process of arterial stiffening and thickening. One study done by Li, Chen, Srinivasan, & Berenson (2004), observed that SBP measured at childhood was an independent predictor of PWV in adulthood. Those with higher SBP levels in childhood (aged 4 – 17 years) had stiffer arteries 26 years later (Li, Chen, Srinivasan, & Berenson, 2004). Based on the findings of this study, psychosocial factors may have an important role in terms of lowering BP in children and preventing further risk factor exposures which would otherwise increase the likelihood of CVD. Possible mechanisms which link stiffness and thickness with BP suggest they are a part of a vicious cycle (Li, Chen, Srinivasan, & Berenson, 2004; O'Rourke, M., Staessen, J., Vlachopoulos, C., Duprez, D., & Plante, G., 2002). This cycle may further explain why in this study we did not see any effect on arterial stiffness or thickness. With an increase in SBP there may be repetitive stress and strain to the arterial wall which results in subsequent arterial stiffness and thickness. The thickness is a result of increased production of vascular smooth muscle cells and collagen, which also translates into an increase in vessel tone/stiffness. This stiffness then leads to further increases in SBP (Schieken, Moskowitz, Bodurtha, Mosteller, Eaves, & Nance, 1988). Therefore, one year may not be enough time to see a significant change in stiffness and thickness. Compliance and distensibility are both defined by a given level of pressure to absolute or relative change in vessel area respectively (O'Rourke, M., Staessen, J., Vlachopoulos, C., Duprez, D., & Plante, G., 2002). PWV and IMT have

shown to be related to SBP in a number of studies (Kim et al., 2007; Jourdan et al., 2005; Poredos, 2004; Schack-Nielson, Molgaard, Larsen, Martyn, & Michaelsen, 2005). While elastic properties of the arterial wall are very much pressure dependent, the length of time it may take to produce a significant visible change in wall composition is not known. In children especially, results may not be evident until later, possibly in adulthood. As for DBP, the reason for not seeing an intervention effect is unclear. However, when discussing arterial stiffness and thickness SBP is suggested to have a substantial prominence over DBP. With a historical perspective quoted in O'Rourke (1990), "SBP represents the maximum force of the heart and so the amount of energy expended by the heart per beat, with other things being equal, varies inversely with the elasticity of the arterial system" (O'Rourke, 1990).

The findings for the current study align well with the literature. For example, McCraty and colleagues (1995), Knapp et al. (1992) and Waldstein et al. (2000) have all reported a decrease in BP after the facilitation of positive emotions (McCraty, Atkinson, Tiller, Rein & Watkins, 1995; Knapp et al., 1992; Waldstein et al., 2000). Higher levels of stress and stress hormones have been found to result in higher levels of BP and to be strong risk factors for the development of hypertension (McEwen & Seeman, 1999; Das & O'Keefe, 2008). Therefore, with the development of coping skills it was proposed that a decrease in BP would be seen. Also, the encouragement of leisure activities provides children with the opportunity to develop new strengths, build on personal resources- including physical resources- and increase positive emotions, all of which may help to decrease BP. However, it is unknown if one unit of the LWM psychosocial intervention had more of an impact on SBP or if it was the interactions between them all that triggered

a positive effect on BP. Nevertheless, the psychosocial intervention did have a positive effect on childhood cardiovascular health within one year, and if exposed for a longer period of time or followed a few years later, it may be possible to see an increase in arterial health as well.

As for the last reported finding of this study it was observed that all three BP groups (HT, PreHT, and NT) in this sample were affected by the intervention in an equivalent fashion. It was suggested that those in the HT group may be exposed to more negative environments and emotions compared to those of the PreHT and NT groups. It was also assumed that those with higher BP levels would have higher baseline values of arterial stiffness and thickness because of their relation to BP. With these propositions, it was thought that the HT group would have a greater improvement after the intervention was implemented compared to the PreHT and NT groups. However, this was not the case in this study. The LWM intervention effect on SBP was not different after taking into account the different BP groups. Participants from each BP status had a similar time x group interaction effect. This finding may be due to the fact that there were no significant differences observed at baseline in any of the stiffness indices between BP groups, and the difference in IMT was very small. Referring back to the vicious cycle, if we did observe more definitive differences in arterial indices at baseline in the HT group we may have seen a greater change in SBP. It is good to note however, that the psychosocial intervention still did improve SBP in children that were normotensive in hopes that it may act as a preventative strategy.

## 6.4 Limitations and Future Considerations

Although the current study adds new knowledge to the literature, there are recognizable limitations that need to be highlighted. First, even though the study design was an intention-to-treat analysis, attendance was not taken of those who were expected to undergo the intervention. Intention-to-treat analysis provides an unbiased assessment of the efficacy of an intervention at the level of adherence observed in the study (Montori & Guyatt, 2001). However, there was no record of how many children actually participated and to what level they participated. If such data were collected, it could have informed the reader about the effectiveness of the intervention at an adherence level expected to be observed in the community. If non-adherence was substantial, then the analysis following intention-to-treat underestimates the magnitude of the treatment effect on SBP that would occur in adherent participants. It would also give an indication of how practical or impractical a LWM psychosocial intervention would be if implemented in schools for children aged 10-13 years. However, although tracking attendance would add to the study it is impractical in a community-level intervention and would detract from the external validity of the study.

Second, the length of the intervention was likely too short for the structural changes being examined in this study to occur. As was determined above, one year may not be enough time to see changes in arterial composition among healthy children. Participants that were included were not known to be clinically diagnosed with any co-morbidity such as obesity or hypertension. Such a clinical sample would make them more at risk and thus more inclined to see improvements. While we did see an improvement in SBP, arterial stiffness and thickness changes can be very small and although dependent

on BP one year may not be enough to visibly see changes in arterial structure and function. Hence, implementing a longer intervention or following up for a longer time period may reveal changes in arterial stiffness and thickness as a result of a reduction in SBP (Schieken, Moskowitz, Bodurtha, Mosteller, Eaves, & Nance, 1988).

Third, automated and manual BP measurements were only measured at a single visit during year one and year two. Repeated visits are necessary for accurate BP classification into one of the three BP groups (HT, PreHT and NT). Pickering et al. (2005) suggests that at least three separates visits are needed to classify children into the correct BP group (Pickering et al., 2005). However, due to the limited access to the elementary schools and having to disrupt class time being a field environment, repeated visits were not possible.

As for future studies, it may be interesting to further analyze whether or not there was a decline in stress and/or negative emotions, an increase in positive emotions or an increase in leisure activities. Although we did observe a LWM psychosocial intervention effect on SBP, it is unknown whether in fact the interrelations between each unit are what contributed to an upward spiral- as hypothesized- eliciting positive emotions and cultivating strengths to improve health. On the other hand, it may have been that one unit/psychosocial factor had a stronger intervention effect compared to the others. Either way, if the change in stress levels, positive or negative emotions, and/or leisure time was examined it may provide a better understanding of the preventative effects psychosocial factors have on BP in children. It may also provide insight on whether or not the LWM is an appropriate base for the development of such an intervention to address cardiovascular health in children. With previous research linking stress levels and positive and negative

emotions to arterial stiffness and thickness in adults (Das & O’Keefe, 2008; Wilkinson et al., 2001; Wilkinson, Franklin, & Cockcroft, 2004; Vlachopoulos et al., 2006), it is suggested that if a decline in stress or increase in positive emotions was found, then exposure for a longer period of time may result in such arterial health changes hypothesized in this study.

Lastly, with a decrease in SBP found within a year of exposure to the psychosocial intervention, it would be of future consideration to study the effects of the LWM psychosocial intervention for a longer period of time. It would be of interest to know if its effects on SBP are sustained over time and if there are noticeable changes in arterial health measures of stiffness and thickness. Furthermore, to better understand the relationship between psychosocial factors and arterial health in children more longitudinal studies are needed to determine the effectiveness of implementing a LWM psychosocial intervention. It is not known how long exposure must be to see an improvement in children’s arterial health and to predict a decrease in cardiovascular complications as adults.

## **6.5 Conclusion**

This study did not show any significant LWM psychosocial intervention effect on arterial health measures of stiffness and /or thickness in children aged 10-13 years contrary to the stated hypotheses. However, it did show a time x group interaction on SBP after controlling for sex, changes in aPHV and changes in BMI. This study suggests that those who were exposed to the psychosocial intervention demonstrated improved cardiovascular health compared to those not exposed. During developmental stages such



an intervention may be a powerful strategy for prevention of disease development and/or progression and if implemented longer may have the potential to improve arterial health as well.

## References

1. Aggoun, Y., Bonnet, D., Sidi, D., Girardet, J. P., Brucker, E., Polak, M., Safar, M., Levy, B. (2000). Arterial mechanical changes in children with familial hypercholesterolemia. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 20, 2070-2075.
2. Ahimastos, A., Formosa, M., Dart, A., & Kingwell, B. (2003). Gender differences in large artery stiffness pre-and post-puberty. *The Journal of Clinical Endocrinology and Metabolism*, 88, 5375-5380.
3. Anderson, D., Metter, E., Hougaku, H., & Najjar, S. (2006). Suppressed anger is associated with increased carotid arterial stiffness in older adults. *American Journal of Hypertension*, 19, 1129-1134.
4. Andersson, B., Mattsson, L., Hahn, L., Marin, P., Lapidus, L., Holm, G., Bengsson, B., Bjorntorp P. (1997). Estrogen replacement therapy decreases hyperandrogenicity and improves glucose homeostasis and plasma lipids in postmenopausal women with noninsulin-dependent diabetes mellitus. *Journal of Clinical Endocrinology and Metabolism*, 82(2), 638-643.
5. Arnett, D., Evans, G., & Riley, W. (1994). Arterial stiffness: A new cardiovascular risk factor? *American Journal of Epidemiology*, 140(8), 669-682.
6. Banach, A., Peralta-Huertas, J., Livingstone, K., Petrella, N., Klentrou, P., Faught, B., Wade, T., O'Leary, D. (2010). Arterial distensibility is reduced in overweight pre- and early pubescent children. *European Journal of Pediatrics*, 169, 695-703.
7. Baskett, J., Lewis, R., Beasley, M., & Gosling, R. (1990). Changes in carotid artery compliance with age. *Age and Ageing*, 19, 241-246.

8. Benetos, A., Waeber, B., Izzo, J., Mitchell, G., Resnick, L., Asmar, R., & Safar, M. (2002). Influence of age, risk factors, and cardiovascular and renal disease on arterial stiffness: Clinical applications. *American Journal of Hypertension*, 15, 1101-1108.
9. Berenson, G. S., Srinivasan, S. R., Bao, W., Newman, W. P., III, & et al. (1998). Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. *The New England Journal of Medicine*, 338(23), 1650-6.
10. Berne, R., & Levy, M. (1997). In Underdown E. (Ed.), *Cardiovascular physiology* (7th ed.) Mosby-Year Book Inc.
11. Berry, K., Cameron, J., Dart, A., Dewar, E., Gatz, C., Jennings, G., Liang, Y., Reid, C., Kingwell, B. (2004). Large artery stiffness contributes to the greater prevalence of systolic hypertension in elderly women. *Journal of the American Geriatrics Society*, 52(3), 368-373.
12. Blacher, J., Guerin, A., Pannier, B., Marchais, S., Safar, M., & London, G. (1999). Impact of aortic stiffness on survival in end-stage renal disease. *Circulation*, 99, 2434-2439.
13. Bruce, M., Beech, B., Sims, M., Brown, T., Wyatt, S., Taylor, H., Williams, D., Crook, E. (2009). Social environmental stressors, psychological factors, and kidney disease. *Journal of Investigative Medicine*, 57(4), 583-589.
14. Bohm, B., Hartmann, K., Buck, M., & Oberhoffer, R. (2009). Sex differences of carotid intima-media thickness in healthy children and adolescents. *Atherosclerosis*, 206, 458-463.

15. Bots, M., Dijk, M., Oren, A., & Grobbee, D. (2002). Carotid intima-media thickness, arterial stiffness and risk of cardiovascular disease: Current evidence. *Journal of Hypertension*, 20(12), 2317-2325.
16. Bots, M., Hoes, A., Koudstaal, P., Hofman, A., & Grobbee, D. (1997). Common carotid intima-media thickness and risk of stroke and myocardial infarction. *Circulation*, 96, 1432-1437.
17. Bradburn, N. (1969). *The structure of psychological well-being*. Chicago: Aldine.
18. Bradburn, N., & Caplovitz, D. (1965). *Reports on happiness: A pilot study of behaviour related to mental health*. Chicago: Aldine.
19. Breslow, L. (1972). A quantitative approach to the world health organization definition of health: Physical, mental and social well-being. *International Journal of Epidemiology*, 1(4), 347-355.
20. Caldwell, L., Darling, N., Payne, L., & Dowdy, B. (1999). "Why are you so bored?" An examination of psychological and social control causes of boredom among adolescents. *Journal of Leisure Research*, 31(2), 103-121.
21. Canadian Society for Exercise Physiology. (2012). Canadian physical activity guidelines and Canadian sedentary behaviour guidelines. Retrieved November, 2012, from <http://www.csep.ca/english/view.asp?x=949>
22. Carnethon, M., Kinder, L., Fair, J., Stafford, R., & Fortmann, S. (2003). Symptoms of depression as a risk factor for incident diabetes: Findings from the national health and nutrition examination epidemiologic follow-up study, 1971–1992. *American Journal of Epidemiology*, 158(5), 416-423.

23. Carruthers, C., & Hood, C. (2007). Building a life of meaning through therapeutic recreation: The leisure and well-being model, part 1. *Therapeutic Recreation Journal*, 41(4), 276-297.
24. Christen, A., Armentano, R., Miranda, A., Graf, S., Santana, D., Zócalo, Y., Baglivo, H., Sánchez, R. (2010). Arterial wall structure and dynamics in type 2 diabetes mellitus methodolgical aspects and pathophysiological findings. *Current Diabetes Reviews*, 6(6), 367-377.
25. Colley, R. C., Garriguet, D., Janssen, I., Craig, C. L., Clarke, J., & Tremblay, M. S. (2011). Physical activity of Canadian adults: Accelerometer results from the 2007 to 2009 Canadian health measures survey. *Health Report*, 22(1), 1-8.
26. Cusi, K., Maezono, K., Osman, A., Pendergrass, M., Patti, M. E., Pratipanawatr, T., DeFronzo, R., Kahn, C., Mandarino, L. J. (2000). Insulin resistance differentially affects the PI 3-kinase- and MAP kinase-mediated signaling in human muscle. *Journal of Clinical Investigation*, 105(3), 311-320.
27. Daniels, S., Khoury, P., & Morrison, J. (2000). Utility of different measures of body fat distribution in children and adolescents. *American Journal of Epidemiology*, 152(12), 1179-1184.
28. Danner, D., Snowdon, D., & Friesen, W. (2001). Positive emotions in early life and longevity: Findings from the nun study. *Journal of Personality and Social Psychology*, 80(5), 804-813.
29. Das, S., & O'Keefe, J. (2008). Behavioral cardiology: Recognizing and addressing the profound impact of psychosocial stress on cardiovascular health. *Current Hypertension Reports*, 10, 374-381.

30. Davidson, K., Jonas, B., Dixon, K., & Markovitz, J. (2000). Do depressive symptoms predict early hypertension incidence in young adults. *Archive of Internal Medicine*, 160, 1495-1500.
31. Depairon, M., Tutta, P., van Melle, G., Hayoz, D., Kappenberger, L., & Darioli, R. (2000). Reference values of intima-media thickness of carotid and femoral arteries in subjects aged 20 to 60 years and without cardiovascular risk factors. *Archives Des Maladies Du Coeur Et Des Vaisseaux*, 93, 721-726.
32. Dernellis, J., & Panaretou, M. (2005). Aortic stiffness is an independent predictor of progression to hypertension in nonhypertensive subjects. *Hypertension*, 45, 426-431.
33. Diez-Roux, A., Nieto, J., Tyroler, H., Crum, L., & Szklo, M. (1995). Social inequalities and atherosclerosis: The atherosclerosis risk in communities study. *American Journal of Epidemiology*, 141(10), 960-972.
34. Dobbelsteyn, C., Joffres, M., MacLean, D., & Flowerdew, G. (2001). A comparative evaluation of waist circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk factors. The Canadian heart health surveys. *International Journal of Obesity*, 25, 652-661.
35. Ellekjær, H., Holmen, J., Indredavik, B., & Terent, A. (1997). Epidemiology of stroke in innherred, norway, 1994 to 1996: Incidence and 30-day case-fatality rate. *Stroke*, 28, 2180-2184.
36. Everson, S., Kaplan, G., Goldberg, D., Salonen, R., & Salonen, J. (1997). Hopelessness and 4-year progression of carotid atherosclerosis. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 17, 1490-1495.

37. Everson-Rose, S., & Lewis, T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review Public Health*, 26, 469-500.
38. Fathi, R., Haluska, B., Isbel, N., Short, L., & Marwick, T. (2004). The relative importance of vascular structure and function in predicting cardiovascular events. *Journal of the American College of Cardiology*, 43(4), 616-623.
39. Fernhall, B., & Agiovlasitis, S. (2008). Arterial function in youth: Window into cardiovascular risk. *Journal of Applied Physiology*, 105, 325-333.
40. Fischer, G., Bashey, R., Rosenbaum, H., & Lyttle, R. (1985). A possible mechanism in arterial wall for mediation of sex difference in atherosclerosis. *Experimental and Molecular Pathology*, 43, 288-296.
41. Fredrickson, B. (1998). What good are positive emotions? *Review of General Psychology*, 2(3), 300-319.
42. Fredrickson, B. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotion. *American Psychology*, 56(3), 218-226.
43. Gardner, A., & Parker, D. (2010). Association between arterial compliance and age in subjects 9-77 years old. *Anigology*, 61(1), 37-41.
44. Gil, T. Y., Sung, C. Y., Shim, S. S., & Hong, Y. M. (2008). Intima-media thickness and pulse wave velocity in hypertensive adolescents. *Journal of Korean Medical Science*, 23, 35-40.
45. Gill, R. W. (1985). Measurement of blood flow by ultrasound: Accuracy and sources of error. *Ultrasound in Medicine and Biology*, 11, 625-641.

46. Glagov, S., Zarins, C., & Giddens, D. P. (1988). Hemodynamics and atherosclerosis. insights and perspectives gained from studies of human arteries. *Archives of Pathology & Laboratory Medicine*, 112(10), 1018-1031.
47. Grobbee, D., & Bots, M. (1994). Carotid artery intima-media thickness as an indication of generalized atherosclerosis. *Journal of Internal Medicine*, 236, 567-573.
48. Hansen, F., Mangell, P., Sonesson, B., & Lanne, T. (1995). Diameter and compliance in the human common carotid artery-variations with age and sex. *Ultrasound in Medicine and Biology*, 21(1), 1-9.
49. Harris, K., & Matthews, K. (2004). Interactions between autonomic nervous system activity and endothelial function: A model for the development of cardiovascular disease. *Psychosomatic Medicine*, 66, 153-164.
50. Hemingway, H., Shipley, M., Mullen, M., Kumari, M., Brunner, E., Taylor, M., Marmot, M. (2003). Social and psychosocial influences on inflammatory markers and vascular function in civil servants (the Whitehall II study). *American Journal of Cardiology*, 92, 984-987.
51. Hood, C., & Carruthers, C. (2007). Enhancing leisure experience and developing resources: The leisure and well-being model, part 2. *Therapeutic Recreation Journal*, 41(4), 298-325.
52. Hoskins, P., Martin, K., & Thrush, A. (Eds.). (2010). *Diagnostic ultrasound: Physics and equipment* (2nd ed.). United States of America, New York: Cambridge University Press.



53. Iannuzzi, A., Licenziati, M., Acampora, C., Salvatore, V., Auriemma, L., Romano, M., Panico, S., Rubba, P., Trevisan, M. (2004). Increased carotid intima-media thickness and stiffness in obese children. *Diabetes Care*, 27(10), 2506-2508.
54. Imholz, B. P., Settels, J. J., van der Meiracker, A. H., Wesseling, K. H., & Wieling, W. (1990). Non-invasive continuous finger BP measurement during orthostatic stress compared to intraarterial pressure. *Cardiovascular Research*, 24(3), 214-221.
55. Ishizu, T., Ishimitsu, T., Yanagi, H., Seo, Y., Obara, K., & Yamaguchi, I. (2004). Effect of age in carotid arterial intima-media thickness in children. *Heart Vessels*, 19, 189-195.
56. Jourdan, C., Wuhl, E., Litwin, M., Fahr, K., Trelewicz, J., Jobs, K., Schenk, J., Grenda, R., Mehls, O., Troger, J., Schaefer, F. (2005). Normative values for intima-media thickness and distensibility of large arteries in healthy adolescents. *Journal of Hypertension*, 23, 1707-1715.
57. Juonala, M., Kahonen, M., Laitinen, T., Hutri-Kahonen, N., Jokinen, E., Taittonen, L., Pietikainen, M., Helenius, H., Viikari, J., Raitakari, O. (2008). Effect of age and sex on carotid intima-media thickness, elasticity and brachial endothelial function in healthy adults: The cardiovascular risk in young Finns study. *European Heart Journal*, 29, 1198-1206.
58. Juonala, M., Jarvisalo, M., Maki-Torkko, N., Kahonen, M., Viikari, J., & Raitakari, O. (2005). Risk factors identified in childhood and decreased carotid artery elasticity in adulthood. The cardiovascular risk in young Finns study. *Circulation*, 112, 1486-1493.

59. Kim, E. J., Park, C. G., Park, J. S., Suh, S. Y., Choi, C. U., Kim, J. W., Kim, S., Lim, H., Rha, S., Seo, H., Oh, D. (2007). Relationship between blood pressure parameters and pulse wave velocity in normotensive and hypertensive subjects: Invasive study. *Journal of Human Hypertension*, 21, 141-148.
60. Knapp, P., Levy, E., Giorgi, R., Black, P., Fox, B., & Heeren, T. (1992). Short-term immunological effects of induced emotion. *Psychosomatic Medicine*, 54, 133-148.
61. Laogun, A., & Gosling, R. (1982). *In vivo* arterial compliance in man. *Clinical Physics and Physiological Measurement*, 3(3), 201-212.
62. Laurent, S. (1995). Arterial wall hypertrophy and stiffness in essential hypertensive patients. *Hypertension*, 26, 355-362.
63. Laurent, S., Girerd, X., Mourad, J., Lacolley, P., Beck, L., Boutouyrie, P., Mignot, J., Safar, M. (1994). Elastic modulus of the radial artery wall material is not increased in patients with essential hypertension. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 14, 1223-1231.
64. Lenard, Z., Studinger, P., Mersich, B., Kocsis, L., & Kollai, M. (2004). Maturation of cardiovagal autonomic function from childhood to young adult age. *Circulation*, 110, 2307-2312.
65. Li, S., Chen, W., Srinivasan, S., & Berenson, G. (2004). Childhood blood pressure as a predictor of arterial stiffness in young adults. The Bogalusa heart study. *Hypertension*, 43, 541-546.
66. Liao, D., Arnett, D., Tyroler, H., Riley, W., Chambless, L., Szklo, M., & Heiss, G. (1999). Arterial stiffness and the development of hypertension: The ARIC study. *Hypertension*, 34, 201-206.

67. Link, B., & Phelan, J. (1995). Social conditions as fundamental causes of disease. *Journal of Health and Social Behaviour*, (Extra Issue), 80-94.
68. Litwin, M., Niemirska, A., Sladowska, J., Wierzbicka, A., Wawer, Z., & Grenda, R. (2006). Left ventricle hypertrophy and arterial wall thickening in children with essential hypertension. *Pediatric Nephrology*, 21, 811-819.
69. Litwin, M., Trelewicz, J., Wawer, Z., Antoniewicz, J., Wierzbicka, A., Rajszyk, P., & Grenda, R. (2004). Intima-media thickness and arterial elasticity in hypertensive children: Controlled study. *Pediatric Nephrology*, 19, 767-774.
70. London, G., & Guerin, A. (1999). Influence of arterial pulse and reflected waves on blood pressure and cardiac function. *American Heart Journal*, 138, S220-S224.
71. London, G., Marchais, S., Guerin, A., & Pannier, B. (2004). Arterial stiffness: Pathophysiology and clinical impact. *Clinical and Experimental Hypertension*, 26(7 & 8), 689-699.
72. Mackenzie, I., Wilkinson, I., & Cockcroft, J. (2002). Assessment of arterial stiffness in clinical practice. *Quarterly Journal of Medicine*, 95, 67-74.
73. McCraty, R., Atkinson, M., Tiller, W., Rein, G., & Watkins, A. (1995). The effects of emotions on short-term power spectrum analysis of heart rate variability. *The American Journal of Cardiology*, 76, 1089-1090.
74. McCrohon, J., Jessup, W., Handelsman, D., & Celermajer, D. (1999). Androgen exposure increases human monocyte adhesion to vascular endothelium and endothelial cell expression of vascular cell adhesion molecule-1. *Circulation*, 99, 2317-2322.

75. McEwen, B., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 896, 30-47.
76. Meyer, A., Kundt, G., Lenschow, U., Schuff-Werner, P., & Kienast, W. (2006). Improvement of early vascular changes and cardiovascular risk factors in obese children after a six-month exercise program. *Journal of the American College of Cardiology*, 48(9), 1865-1870.
77. Midei, A., & Matthews, K. (2009). Social relationships and negative emotional traits are associated with central adiposity and arterial stiffness in healthy adolescents. *Health Psychology*, 28(3), 347-353.
78. Mirwald, R., Baxter-Jones, A., Bailey, D., & Beunen, G. (2002). An assessment of maturity from anthropometric measurements. *Medicine and Science in Sports and Exercise*, 34(4), 689-694.
79. Montori, V., & Guyatt, G. (2001). Intention-to-treat principle. *Canadian Medical Association Journals*, 165(10), 1339-1341.
80. Muller-Delp, J. (2011). Age-induced endothelial dysfunction: Is it time to redefine the “reactivity” of reactive oxygen species? *Journal of Applied Physiology*, 110, 1152-1153.
81. Nichols, W., & O'Rourke, M. (2005). In Koster J., Burrows S. and Wilkinson N. (Eds.), *McDonald's blood flow in arteries* (5th ed.). Great Britain: Hodder Arnold.
82. Nickenig, G., Röling, J., Strehlow, K., Schnabel, P., & Böhm, M. (1998). Insulin induces upregulation of vascular AT1 receptor gene expression by posttranscriptional mechanisms. *Circulation*, 98, 2453-2460.

83. Oliver, J., & Webb, D. (2003). Non-invasive assessment of arterial stiffness and risk of atherosclerotic events. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 23, 554-566.
84. O' Rourke, M. (1990). Arterial stiffness, systolic blood pressure, and logical treatment of arterial hypertension. *Hypertension*, 15, 339-347.
85. O'Rourke, M., & Mancia, G. (1999). Arterial stiffness. *Journal of Hypertension*, 17, 1-4.
86. O'Rourke, M., Staessen, J., Vlachopoulos, C., Duprez, D., & Plante, G. (2002). Clinical applications of arterial stiffness; definitions and reference values. *American Journal of Hypertension*, 15, 426-444.
87. Paterniti, S., Zureik, M., Ducimetière, P., Touboul, P., Fève, J., & Alperovitch, A. (2001). Sustained anxiety and 4-year progression of carotid atherosclerosis. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 21, 136-141.
88. Phillips, A. (2009). *The association between blood pressure and vascular characteristics in children* (Unpublished Masters of Science). Brock University, St. Catharines, Ontario.
89. Pickering, T., Hall, J., Appel, L., Falkner, B., Graves, J., Hill, M., Jones, D., Kurtz, T., Sheps, S., Roccella, E. (2005). Recommendations for blood pressure measurement in humans and experimental animals: Part 1: Blood pressure measurement in humans: A statement for professionals from the subcommittee of professional and public education of the American heart association council on high blood pressure research. *Circulation*, 111, 697-716.

90. Poredos, P. (2004). Intima-media thickness: Indicator of cardiovascular risk and measure of the extent of atherosclerosis. *Vascular Medicine*, 9, 46-54.
91. Raitakari, O., Juonala, M., Kahonen, M., Taittonen, L., Laitinen, T., Maki-Torkko, N., Jarvisalo, M., Uhari, M., Jokinen, E., Ronnema, T., Akerblom, H., Viikari, J. (2003). Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood. the cardiovascular risk in young Finns study. *Journal of American Medical Association*, 290, 2277-2283.
92. Rajagopalan, S., Brook, R., Rubenfire, M., Pitt, E., Young, E., & Pitt, B. (2001). Abnormal brachial artery flow-mediated vasodilation in young adults with major depression. *The American Journal of Cardiology*, 88, 196-198.
93. Reed, K., Warburton, D., Lewanczuk, R., Haykowsky, M., Scott, J., Whitney, C., McGavock, J., McKay, H. (2005). Arterial compliance in young children: The role of aerobic fitness. *European Journal of Cardiovascular Prevention and Rehabilitation*, 12, 492-497.
94. Resnick, L., Militianu, D., Cunnings, A., Pipe, J., Evelhoch, J., & Soulen, R. (1997). Direct magnetic resonance determination of distensibility in essential hypertension. *Hypertension*, 30, 654-659.
95. Richman, L., Kubzansky, L., Maselko, J., Kawachii, I., Choo, P., & Bauer, M. (2005). Positive emotion and health: Going beyond the negative. *Health Psychology*, 24(4), 422-429.
96. Riggio, S., Mandraffino, G., Sardo, M., Iudicello, R., Camarda, N., Imbalzano, E., Alibrandi, A., Saitta, C., Carerj, S., Arrigo, T., Saitta, A. (2010). Pulse wave velocity and augmentation index but not intima-media thickness, are early indicators of

- vascular damage in hypercholesterolemic children. *European Journal of Clinical Investigation*, 40(3), 250-257.
97. Riley, W., Freedman, D., Higgs, N., Barnes, R., Zinkgraf, S., & Berenson, G. (1986). Decreased arterial elasticity associated with cardiovascular disease risk factors in the young. Bogalusa heart study. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 6, 378-386.
  98. Rowe, J., & Kahn, R. (1987). Human aging: Usual and successful. *Science New Series*, 237(4811), 143-149.
  99. Safar, M., Levy, B., & Struijker Boudier, H. (2003). Current perspectives on arterial stiffness and pulse pressure in hypertension and cardiovascular diseases. *Circulation*, 107, 2864-2869.
  100. Sakuragi, S., Abhayaratna, K., Gravenmaker, K., O'Reilly, C., Sriksalanukul, W., Budge, M., Telford, R., Abhayaratna, W. (2009). Influence of adiposity and physical activity on arterial stiffness in healthy children. The lifestyle of our kids study. *Hypertension*, 53, 611-616.
  101. Salovey, P., Rothman, A., Detweiler, J., & Steward, W. (2000). Emotional states and physical health. *American Psychologist*, 55, 110-121.
  102. Sass, C., Herbeth, B., Chapet, O., Siest, G., Visvikis, S., & Zannad, F. (1998). Intima-media thickness and diameter of carotid and femoral arteries in children, adolescents and adults from the Stanislas cohort: Effect of age, sex anthropometry and blood pressure. *Journal of Hypertension*, 16, 1593-1602.

103. Schack-Nielsen, L., Molgaard, C., Larsen, D., Martyn, C., & Michaelsen, K.  
(2005). Arterial stiffness in 10-year-old children: Current and early determinants.  
*British Journal of Nutrition*, 94, 1004-1011.
104. Schieken, R. M., Moskowitz, W. B., Bodurtha, J., Mosteller, M., Eaves, L., &  
Nance, W. (1988). Aortic stiffness: A new doppler echocardiographic measure  
predictive of systolic blood pressure in children. *Journal of American College of  
Cardiology*, 11, 1297-1300.
105. Schiffrin, E. (2004). Vascular stiffening and arterial compliance. implications for  
systolic blood pressure. *American Journal of Hypertension*, 17, 395-485.
106. Schneiderman, N., Antoni, M., Saab, P., & Ironson, G. (2001). Health psychology:  
Psychosocial and behavioural aspects of chronic disease management. *Annual  
Review of Psychology*, 52, 555-580.
107. Senzaki, H., Akagi, M., Hishi, T., Ishizawa, A., Yanagisawa, M., Masutani, S.,  
Kobayashi, T., Awa, S. (2002). Age-associated changes in arterial elastic properties  
in children. *European Journal of Pediatrics*, 161, 547-551.
108. Shonkoff, J., Boyce, W., & McEwen, B. (2009). Neuroscience, molecular biology,  
and the childhood roots of health disparities. Building a new framework for health  
promotion and disease prevention. *Journal of the American Medical Association*,  
301(21), 2252-2259.
109. Simon, T., Boutouyrie, P., Simon, J. M., Laloux, B., Tournigand, C., Tropeano, A.  
I., Laurent, S., Jaillon, P. (2002). Influence of tamoxifen on carotid intima-media  
thickness in postmenopausal women. *Circulation*, 106, 2925-2929.



110. Smith, E. (2006). The strength-based counselling model. *The Counselling Psychologist*, 34(1), 13-79.
111. Sorensen, K., Celermajer, D., Georgakopoulos, D., Hatcher, G., Betteridge, D. J., & Deanfield, J. (1994). Impairment of endothelium-dependent dilation is an early event in children with familial hypercholesterolemia and is related to the lipoprotein (a) level. *Clinical Journal of Investigation*, 93(1), 50-55.
112. Sorof, J., Alexandrov, A., Cardwell, G., & Portman, R. (2003). Carotid artery intima-media thickness and left ventricular hypertrophy in children with elevated blood pressure. *Pediatrics*, 111, 61-66.
113. Srinivasan, S., Bao, W., Wattigney, W., & Berenson, G. (1996). Adolescent overweight is associated with adult overweight and multiple cardiovascular risk factors: The bogalusa heart study. *Metabolism: Clinical and Experimental*, 45(2), 235-240.
114. Stabouli, S., Kotsis, V., Karagianni, C., Zakopoulos, N., & Konstantopoulous, A. (2012). Blood pressure and carotid artery intima-media thickness in children and adolescents: The role of obesity. *Hellenic Journal of Cardiology*, 53, 41-47.
115. Steptoe, A., Wardle, J., & Marmot, M. (2005). Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. *Proceedings of the National Academy of Sciences*, 102(18), 6508-6512.
116. Strike, P., & Steptoe, A. (2004). Psychosocial factors in the development of coronary artery disease. *Progress in Cardiovascular Diseases*, 46(4), 337-347.
117. Sullivan, M., Wood, L., Terry, J., Brantley, J., Charles, A., McGee, V., Johnson, D., Krucoff, M., Rosenberg, B., Bosworth, H., Adams, K., Cuffe, M. (2009). The

- support, education, and research in chronic heart failure study (SEARCH): A mindfulness-based psychoeducational intervention improves depression and clinical symptoms in patients with chronic heart failure. *American Heart Journal*, 157(1), 84-90.
118. Tanaka, H., Dinunno, F., Monahan, K., Clevenger, C., DeSouza, C., & Seals, D. (2000). Aging, habitual exercise, and dynamic arterial compliance. *Circulation*, 102, 1270-1275.
  119. Tanaka, H., & Safar, M. (2005). Influence of lifestyle modifications on arterial stiffness and wave reflections. *American Journal of Hypertension*, 18, 137-144.
  120. Taylor, R., Jones, I., Williams, S., & Goulding, A. (2000). Evaluation of waist circumference, waist-to-hip ratio, and the conicity index as screening tools for high trunk fat mass, as measured by dual-energy X-ray absorptiometry, in children aged 3–19 y. *The American Journal of Clinical Nutrition*, 72, 490-495.
  121. Torrance, B., McGuire, K., Lewanczuk, R., & McGavock, J. (2007). Overweight, physical activity and high blood pressure in children: A review of the literature. *Vascular Health and Risk Management*, 3(1), 139-149.
  122. Tounian, P., Aggoun, Y., Dubem, B., Varille, V., Guy-Grand, B., Girardet, J., & Bonnet, D. (2001). Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: A prospective study. *The Lancet*, 358, 1400-1404.
  123. Tremollieres, F., Cigagna, F., Alquier, C., Cauneille, C., Pouilles, J., & Ribot, C. (2000). Effect of hormone replacement therapy on age-related increase in carotid artery intima-media thickness in postmenopausal women. *Atherosclerosis*, 153, 81-88.

124. Urbina, E., Alpert, B., Flynn, J., Hayman, L., Harshfield, G., Jacobson, M., Mahoney, L., McCrindle, B., Mietus-Synder, M., Steinberger, J., Daniels, S. (2008). Ambulatory blood pressure monitoring in children and adolescents: Recommendations for standard assessment: A scientific statement from the American heart association atherosclerosis, hypertension, and obesity in youth committee of the council on cardiovascular disease in the young and the council for high blood pressure research. *Hypertension*, 52, 433-451.
125. Urbina, E., Khoury, P., McCoy, C., Daniels, S., Kimball, T., & Dolan, L. (2011). Cardiac and vascular consequences of pre-hypertension in youth. *Journal of Clinical Hypertension (Greenwich)*, 13(5), 332-342.
126. Urbina, E. M., Kimball, T. R., McCoy, C. E., Khoury, P. R., Daniels, S. R., & Dolan, L. M. (2009). Youth with obesity and obesity-related type 2 diabetes demonstrate abnormalities in carotid structure and function. *Circulation*, 119(22), 2913-2919.
127. Van Bortel, L., Vanmolkot, F., Van der Heijden-Spek, J., Bregu, M., Staessen, J., & Hoeks, A. (2001). Does B-mode common carotid artery intima-media thickness differ from M-mode? *Ultrasound in Medicine and Biology*, 27(10), 1333-1336.
128. van der Heijden-Spek, J., Staessen, J., Fagard, R., Hoeks, A., Struijker Boudier, H., & Van Bortel, L. (2000). Effect of age on brachial artery wall properties differs from the aorta and is gender dependent : A population study. *Hypertension*, 35, 637-642.
129. Van Merode, T., Hick, P., Hoeks, A., & Reneman, R. (1989). Non-invasive assessment of artery wall properties in children aged 4-19 years. *Pediatric Research*, 25, 94-96.

130. Vargas, R., Wroblewska, B., Rego, A., Hatch, J., & Ramwell, P. (1993). Oestradiol inhibits smooth muscle cell proliferation of pig coronary artery. *British Journal of Pharmacology*, 109, 612-617.
131. Vercoza, A., Baldisserotto, M., Abaete de los Santos, Eduardo Poli-de-Figueiredo, C., & Otavio d'Avila, D. (2009). Cardiovascular risk factors and carotid intima-media thickness in asymptomatic children. *Pediatric Cardiology*.
132. Vlachopoulos, C., Kosmopoulou, F., Alexopoulos, N., Loakeimidis, N., Siasos, G., & Stefanadis, C. (2006). Acute mental stress has a prolonged unfavourable effect on arterial stiffness and wave reflections. *Psychosomatic Medicine*, 68, 231-237.
133. Vlachopoulos, C., Xaplanteris, P., Alexopoulos, N., Aznaouridis, K., Vasiliadou, C., Baou, K., Stefanadi, E., Stefanadis, C. (2009). Divergent effects of laughter and mental stress on arterial stiffness and central hemodynamics. *Psychosomatic Medicine*, 71, 446-453.
134. Waldstein, S., Kop, W., Schmidt, L., Haufler, A., Krantz, D., & Fox, N. (2000). Frontal electrocortical and cardiovascular reactivity during happiness and anger. *Biological Psychology*, 55, 3-23.
135. Waller, B., Orr, C., Slack, J., Pinkerton, C., Van Tassel, J., & Peters, T. (1992). Clinical pathologic correlations. Anatomy, histology, and pathology of coronary arteries: A review relevant to new interventional and imaging techniques-part 1. *Clinical Cardiology*, 15, 451-457.
136. Wendelhag, I., Gustavsson, T., Suurkula, M., Berglund, G., & Wikstrand, J. (1991). Ultrasound measurement of wall thickness in the carotid artery: Fundamental

- principles and description of a computerized analysing system. *Clinical Physiology*, 11, 565-577.
137. Westendorp, I., Bots, M., Grobbee, D., Reneman, R., Hoeks, A., Van Popele, N., Hofman, A., Witteman, J. (1999). Menopausal status and distensibility of the common carotid artery. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 19, 713-717.
  138. White, R., & Hollier, L. (Eds.). (2005). *Vascular surgery: Basic science and clinical correlations* (2nd ed.) Blackwell Publishing.
  139. Wiegman, A., de Groot, E., Hutten, B., Rodenburg, J., Gort, J., Bakker, H., Sijbrands, E., Kastelein, J. (2004). Arterial intima-media thickness in children heterozygous for familial hypercholesterolaemia. *Lancet*, 363, 369-370.
  140. Wildman, R., Farhat, G., Patel, A., Mackey, R., Brockwell, S., Thompson, T., & Sutton-Tyrrell, K. (2005). Weight change is associated with change in arterial stiffness among healthy young adults. *Hypertension*, 45, 187-192.
  141. Wildman, R., Mackey, R., Bostom, A., Thompson, T., & Sutton-Tyrrell, K. (2003). Measures of obesity are associated with vascular stiffness in young and older adults. *Hypertension*, 42, 468-473.
  142. Wilkinson, I., MacCallum, H., Hupperetz, P., van Thoor, C., Cockcroft, J., & Webb, D. (2001). Changes in the derived central pressure waveform and pulse pressure in response to angiotensin II and noradrenaline in man. *Journal of Physiology*, 530.3, 541-550.

143. Wilkinson, I., Franklin, S., & Cockcroft, J. (2004). Nitric oxide and the regulation of large artery stiffness: From physiology to pharmacology. *Hypertension*, 44, 112-116.
144. Williams, J., Din-Dzietham, R., & Szklo, M. (2006). Trait anger and arterial stiffness: Results from the atherosclerosis risk in communities (ARIC) study. *Preventative Cardiology*, 9, 14-20.
145. Wilson, B. (2006). Cardiovascular imaging using CT and MR. *Radiologic Technology*, 77(6), 461-474.
146. Woo, K., Chook, P., Yu, C., Sung, R., Qiao, M., Leung, S., Lam, C., Metreweli, C., Celermajer, D. (2004). Effects of diet and exercise on obesity-related vascular dysfunction in children. *Circulation*, 109, 1981-1986.
147. Yan, L., Liu, K., Matthews, K., Daviglus, M., Ferguson, T., & Kiefe, C. (2003). Psychosocial factors and risk of hypertension. The coronary artery risk development in young adults (CARDIA) study. *Journal of the American Medical Association*, 290(16), 2138-248.
148. Zieman, S., Melenovsky, V., & Kass, D. (2005). Mechanisms, pathophysiology, and therapy of arterial stiffness. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 25, 932-943.
149. Zizek, B., & Poredos, P. (2002). Dependence of morphological changes of the carotid arteries on essential hypertension and accompanying risk factors. *International Journal of Angiology*, 21, 70-77.
150. Zureik, M., Touboul, P., Bonithon-Kopp, C., Courbon, D., Berr, C., Leroux, C., & Ducimetie`re, P. (1999). Cross-sectional and 4-year longitudinal associations

between brachial pulse pressure and common carotid intima-media thickness in a general population: The EVA study. *Stroke*, 30, 550-555.

## Appendix A

### Ethics Approval Form



Brock University  
Research Ethics Board  
Tel: 905-888-6660 ext. 3036  
Email: reb@brocku.ca

#### Certificate of Ethics Clearance for Human Participant Research

DATE: November 17, 2010  
PRINCIPAL INVESTIGATOR: WADE, Terrance - Community Health Sciences  
FILE: 06-315 - WADE  
TYPE: Masters Thesis/Project STUDENT:  
SUPERVISOR:  
TITLE: Social Determinants of Child Hypertension

#### ETHICS CLEARANCE GRANTED

Type of Clearance: MODIFICATION Expiry Date: 6/30/2011

The Brock University Research Ethics Board has reviewed the above named research proposal and considers the procedures, as described by the applicant, to conform to the University's ethical standards and the Tri-Council Policy Statement. Clearance granted from 11/17/2010 to 6/30/2011.

The Tri-Council Policy Statement requires that ongoing research be monitored by, at a minimum, an annual report. Should your project extend beyond the expiry date, you are required to submit a Renewal form before 6/30/2011. Continued clearance is contingent on timely submission of reports.

To comply with the Tri-Council Policy Statement, you must also submit a final report upon completion of your project. All report forms can be found on the Research Ethics web page.

In addition, throughout your research, you must report promptly to the REB:

- Changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
- All adverse and/or unanticipated experiences or events that may have real or potential unfavourable implications for participants;
- New information that may adversely affect the safety of the participants or the conduct of the study;
- Any changes in your source of funding or new funding to a previously unfunded project.

We wish you success with your research.

Approved:

Michelle McGinn, Chair  
Research Ethics Board (REB)

**Note:** Brock University is accountable for the research carried out in its own jurisdiction or under its auspices and may refuse certain research even though the REB has found it ethically acceptable.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of research at that site.



## Appendix B Informed Consent Form

\*\*\*staple ballot to forms!\*\*\*



Dear Parents and Guardians:

Did you know that adults in Canada and the United States have a 90% chance of developing high blood pressure (also known as hypertension)? Did you also know that high blood pressure is one of the strongest predictors of heart disease? Some scientists even suggest that high blood pressure can start in childhood! Luckily, the Heart and Stroke Foundation of Ontario has provided us with a great opportunity to look at high blood pressure and heart health in children in the Niagara Region.

With the help of the Heart and Stroke Foundation, we have organized a research team to help gain a better understanding of high blood pressure among children. We already looked at students in the NCDSB in 2007/2008, but now we are asking for your help. We would like to invite you and your child to join our team and help us fight childhood high blood pressure – all it takes is a few blood pressure readings and some questions answered! Through our previous work, we have learned some of the factors that contribute to childhood high blood pressure, and now we would like to offer suggestions to families, schools, and communities to improve child heart health. By joining our efforts, this knowledge could help all children lead healthier lives and reduce their chance of having heart disease as adults.

Not only will you have the opportunity to learn your child's blood pressure and body mass, but you will gain a better understanding of their heart health, and have the chance to win prizes! Please read the attached description for more information on our study.

Please return the signed consent form to the teacher.

<b>CONSENT FORM</b>		
Student Name:		School:
Parent/Guardian Name:		Teacher: Grade:
Home Address:		
City/Town:	Postal Code:	Home Phone: ( ) -
<input type="checkbox"/> I do give permission for my child to participate in this stage of the Brock University HBeat study conducted by Dr. Terrance J. Wade.		
<input type="checkbox"/> I do not give permission for my child to participate in this stage of the Brock University HBeat study conducted by Dr. Terrance J. Wade.		
Signature of Parent/Guardian:		Date:
Signature of Student:		Date:

Additionally, all data collected may be used later to answer other research questions that may arise from this study. We would like your permission to keep the information that you and your child provided on file after this research study is over. All stored personal data will be kept strictly confidential and all information will be coded so that no one will be able to identify you or your child. At any time, you can ask to have your information removed and not included in any future projects by contacting Dr. Terrance J. Wade (905-688-5550 ext 4146). The investigators reserve the right to withdraw your child from the study if they believe that circumstances have arisen which warrant doing so.

<input type="checkbox"/> I do give permission to have my information and my child's information stored to use to answer future research questions after the HBeat study is over.	
<input type="checkbox"/> I do not give permission to have my information and my child's information stored to use to answer future research questions after the HBeat study is over.	
Signature of Parent/Guardian:	Date:
Signature of Student:	Date:

Please have your child return this form to their classroom teacher.

**\*\*Please keep this information sheet for your records\*\***



HBeat stands for the Heart Behavioural and Environmental Assessment Team. The team examines how children's activities, surroundings, and actions affect their blood pressure. During our first Phase in 2007/2008, we examined all these factors in 1,900 students' lives, and have discovered some things that contribute to a rise in blood pressure. Now we are beginning our second Phase - from March 2010 to May 2010, we will test approximately 1,000 students in grades 5, 6 and 7. We will then follow up with these students again in 2011 when they are in grades 6, 7 and 8.

By saying yes, you and your child will be helping our study immensely. To begin, we will come to the school and measure your child's blood pressure, height, weight, hip circumference, and waist circumference, and give them a questionnaire about their activities. These measurements will take about 30 minutes of class time. We will also send home a questionnaire that you, the parent/guardian, will complete and return, and one for your child to complete and return. The parent form contains questions about yourself, your family and child(ren), your home, and your neighbourhood. The child form asks about their experiences, how they feel, and what they like to do and not do. In addition, one week later, we will randomly select about 15 students from each school to have a more detailed look at his/her heart and arteries (please see the attached information sheet for a more detailed outline of this further testing). This testing will be one-on-one, and will last approximately 30mins to 1hour. Parents are more than welcome to be present for any and all testing.

Following this initial testing, we will return to your school in one year (2011), and go through these procedures again.

In addition to the benefits of learning about your child's heart health, you and your child will have an opportunity to **\*WIN\* a family get-away to Great Wolf Lodge!** Every school involved will have their own draw, and one family from each school will win! This get-away includes a two-day water park pass, evening accommodation, and food vouchers for dinner and breakfast, for all immediate members of a family. Every student in each grade 5, 6, and 7 class has a chance to win! In addition, with each further portion of our study that you complete, you will receive an additional ballot into the draw! For example, a family will have one ballot for having a child in grade 5, 6, or 7. The family will receive another ballot when they return the completed consent form (positive or negative consent!). The family will receive another ballot when the child participates in our blood pressure testing, another for returning the completed child questionnaire, and another for completing and returning the parent questionnaire! That's 5 ballots that you can possibly enter in the draw, and 5 chances of winning!!!

Your involvement and your child's involvement is completely voluntary and you both may withdraw from the study at any time. There is no obligation for you or your child to answer any or all of these questions, or to take part in any aspect of this project. If you do choose to withdraw, your prize ballots will remain in the draw.

*\* This study has been reviewed and approved by the Research Ethics Boards from both Brock University and the Niagara Catholic District School Board.*

*\*Any information about you and your child will only be seen by the researchers involved in this project. All private information will be kept strictly confidential and coded anonymously so that your name and your child's name are not associated with any answers to any questions. When the results of this project are presented, we will only use the combined data from all families so that no single child or family can be identified.*

*\*When the first stage of Phase 2 is complete, June 2010, we will send home your child's blood pressure results. We will do this again after the 2011 follow-up testing. When the entire Phase 2 is complete, we will provide your school with a report that you will be able to view. We also hope to publish these collective findings in peer-reviewed scientific journals. Please contact the researchers directly if you wish to personally obtain this information.*

We will be very grateful if you agree to take part in this important study. If you would like more information about the HBeat program, or if you have any questions, please contact Dr. Terrance Wade.

Thank you for your help!

<signature>

Dr. Terrance J. Wade

**\*\*Please keep this information sheet for your records\*\***



## **Detailed Testing**

If your child is invited to participate in the second, more detailed testing one week after we test their blood pressure, below is an outline of the details. All measure will be taken in a private room by qualified individuals, and no risk is involved.

In addition to the blood pressure, weight, height, hip and waist measurements one week earlier, your child will have their skinfold thickness measured. Skinfold thickness will be assessed using a non-invasive method that measures skin thickness. The researcher lightly pinches the skin at the appropriate site to raise a double layer of skin and the underlying adipose tissue, but not the muscle. The calipers are then applied at right angles to the pinch and a reading is taken. Skinfold measures will be taken at two sites including the subscapular (lower shoulder blade) and triceps (back of the upper arm).

Your child will also lie down and have their heart rate monitored while their heart and right carotid artery (artery in the neck) are imaged using Doppler ultrasound (the same type of ultrasound seen in a hospital). Heart rate will be measured using sensors placed on the skin of your child's upper chest. These sensors are electrodes used to detect the electrical activity generated by the heart and do not transmit electrical signals into the body from the heart rate monitor. All carotid artery and heart ultrasound measures will be taken in a lying position. The carotid artery ultrasound will be performed using a small transducer to visualize the carotid artery. As well, carotid artery blood pressure will be taken at the same time using a thin pen-like device that is lightly pressed against the neck. Both the probe and pen-like-device will be pressed against the neck on opposite sides. It is a non-invasive procedure. Second, the ultrasound of the heart will be performed with a small probe placed between your child's ribs on the left side of their chest. This procedure is also non-invasive and no risk is involved.

Parents are more than welcome to be present for testing.

### **Potential Risks and Discomforts**

No risk is involved with these procedures. If an injury occurs at any time during the investigation, appropriate first aid/CPR will be administered and you will be advised to seek necessary medical help.

### **Benefits**

A potential benefit for your child's participation in this project is the knowledge of their blood pressure, heart and artery assessment, as well as any underlying cardiovascular disease risk factors (take out??? Jose usually did this).

If you have any further questions regarding your rights as a research participant contact the Research Ethics Officer in the Office of Research Services at (905) 688-5550 x3035 or email at reb@brocku.ca.

## Appendix C

### Blood Pressure Percentile Table

Sex	Age	Height Percentile	Index	Systolic			Diastolic		
				Mean	p90	p95	Mean	p90	p95
F	10	all	1100	92.7	107.5	107.7	57.3	70.3	74.0
F	11	5th	1111	91.2	98.7	101.0	57.5	65.3	77.7
F	11	10th	1112	90.7	102.7	102.7	56.3	70.7	71.7
F	11	25th	1113	88.1	96.0	100.3	53.9	65.0	71.3
F	11	50th	1114	92.6	106.3	112.0	56.4	68.7	72.5
F	11	75th	1115	92.4	101.7	104.0	57.1	64.3	68.3
F	11	90th	1116	91.1	97.0	107.5	56.7	63.0	67.7
F	11	95th	1117	94.2	104.0	106.3	60.4	72.0	72.7
F	12	5th	1121	92.7	104.7	120.7	57.8	71.7	86.0
F	12	10th	1122	93.0	102.3	105.0	56.2	67.0	71.3
F	12	25th	1123	91.6	102.0	105.7	56.8	68.0	69.3
F	12	50th	1124	92.2	102.7	107.7	56.3	68.0	71.7
F	12	75th	1125	93.9	104.7	110.3	57.0	66.7	73.0
F	12	90th	1126	93.3	103.7	110.7	57.3	67.7	70.3
F	12	95th	1127	98.8	111.0	127.3	62.0	74.0	79.0
F	13	5th	1131	89.6	106.0	106.0	54.4	71.7	71.7
F	13	10th	1132	92.6	98.7	116.0	57.4	65.3	67.7
F	13	25th	1133	95.1	105.3	116.0	58.9	72.3	76.7
F	13	50th	1134	92.0	102.3	103.3	57.0	65.3	66.3
F	13	75th	1135	93.0	104.7	106.0	57.5	68.7	71.7
F	13	90th	1136	94.5	103.3	106.0	57.9	65.0	70.3
F	13	95th	1137	95.2	109.3	113.0	59.5	68.7	70.7
F	14	all	1140	94.3	106.0	107.5	59.3	67.3	68.7
M	10	all	2100	92.6	101.7	103.3	60.2	69.0	70.7
M	11	5th	2111	88.0	100.7	100.7	53.9	69.3	69.3
M	11	10th	2112	92.5	102.3	108.0	58.4	70.0	75.7
M	11	25th	2113	92.4	105.0	111.7	58.0	75.0	77.5
M	11	50th	2114	90.4	97.7	99.7	55.5	63.7	71.3
M	11	75th	2115	91.7	103.7	107.0	55.9	65.0	69.0
M	11	90th	2116	92.1	101.7	111.2	55.1	63.5	72.2
M	11	95th	2117	95.0	109.7	112.0	58.0	74.7	77.5
M	12	5th	2121	92.6	102.7	102.7	58.3	72.3	72.3
M	12	10th	2122	91.7	100.3	113.0	55.8	68.3	78.0
M	12	25th	2123	93.7	107.5	117.5	57.9	70.0	70.0
M	12	50th	2124	92.9	105.7	115.0	56.8	65.3	67.7
M	12	75th	2125	92.0	102.3	105.0	55.6	64.3	67.7
M	12	90th	2126	93.7	103.7	104.7	55.2	65.3	66.3
M	12	95th	2127	97.0	110.0	113.7	58.9	68.3	70.3
M	13	5th	2131	91.3	97.3	97.3	55.1	65.3	65.3
M	13	10th	2132	93.9	108.8	116.5	58.3	73.3	80.5
M	13	25th	2133	91.9	102.0	107.3	54.8	62.7	69.3
M	13	50th	2134	93.2	105.2	108.5	57.4	66.5	70.0
M	13	75th	2135	96.4	106.7	107.5	55.7	64.3	65.0
M	13	90th	2136	96.5	108.0	113.0	57.4	65.7	69.7
M	13	95th	2137	96.8	104.7	115.3	58.4	66.0	73.3
M	14	all	2140	93.5	104.0	105.7	56.5	62.0	65.3

## Appendix D

### Data Collection Sheets



#### STUDENT DATA COLLECTION FORM

Student: \_\_\_\_\_

School: \_\_\_\_\_

Grade: \_\_\_\_\_

Teacher: \_\_\_\_\_

☐ Male☐ FemaleDate: \_\_\_\_ / \_\_\_\_ / \_\_\_\_  
(mm / dd / yy)

Time: \_\_\_\_ : \_\_\_\_ am/pm

☐ Errors Occurred☐ Manual

Questionnaire checked: \_\_\_\_ (RA initial)

Reading:						Manual Readings	
	2nd	3rd	4th	5th	6th	1st	2nd
Systolic							
Diastolic							
Heart Rate							

Recorded by: \_\_\_\_ (RA initial)

Measurements:	1st	2nd	3rd
Height (cm)			
Weight (kg)			
Waist Girth (cm)			
Hip Girth (cm)			

Physical Measurements by: \_\_\_\_ (RA initial)



Subject ID Number: \_\_\_\_\_

Image Number: \_\_\_\_\_

Beat Number: \_\_\_\_\_

**Diastole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

**Systole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

Image Number: \_\_\_\_\_

Beat Number: \_\_\_\_\_

**Diastole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

**Systole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

Image Number: \_\_\_\_\_

Beat Number: \_\_\_\_\_

**Diastole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

**Systole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

Beat Number: \_\_\_\_\_

**Diastole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

**Systole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

Beat Number: \_\_\_\_\_

**Diastole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

**Systole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

Beat Number: \_\_\_\_\_

**Diastole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

**Systole**

Diameter 1: \_\_\_\_\_

Diameter 2: \_\_\_\_\_

Diameter 3: \_\_\_\_\_

Notes/Comments: \_\_\_\_\_

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

**Subject ID Number:** \_\_\_\_\_

**Image Number:** \_\_\_\_\_

**Beat Number:** \_\_\_\_\_

**End-Diastole**

IMT Average: \_\_\_\_\_

IMT Maximum: \_\_\_\_\_

IMT Mean: \_\_\_\_\_

IMT PTS: \_\_\_\_\_

**Beat Number:** \_\_\_\_\_

**End-Diastole**

IMT Average: \_\_\_\_\_

IMT Maximum: \_\_\_\_\_

IMT Mean: \_\_\_\_\_

IMT PTS: \_\_\_\_\_

**Image Number:** \_\_\_\_\_

**Beat Number:** \_\_\_\_\_

**End-Diastole**

IMT Average: \_\_\_\_\_

IMT Maximum: \_\_\_\_\_

IMT Mean: \_\_\_\_\_

IMT PTS: \_\_\_\_\_

**Beat Number:** \_\_\_\_\_

**End-Diastole**

IMT Average: \_\_\_\_\_

IMT Maximum: \_\_\_\_\_

IMT Mean: \_\_\_\_\_

IMT PTS: \_\_\_\_\_

**Image Number:** \_\_\_\_\_

**Beat Number:** \_\_\_\_\_

**End-Diastole**

IMT Average: \_\_\_\_\_

IMT Maximum: \_\_\_\_\_

IMT Mean: \_\_\_\_\_

IMT PTS: \_\_\_\_\_

**End-Diastole**

IMT Average: \_\_\_\_\_

IMT Maximum: \_\_\_\_\_

IMT Mean: \_\_\_\_\_

IMT PTS: \_\_\_\_\_

<b>Notes/Comments:</b>

## Appendix E

### Learning Activities for LWM Intervention

#### The Good Stuff

Purpose: To learn what makes you happy, and to know that you can change how you're feeling, just by paying attention to *the good stuff!*

Time: 10 minutes

Procedure:

- 1) Let the Health Club members go to morning recess, just like they normally would
- 2) Before afternoon recess, ask them to pay attention to:  
What makes them feel happy? What do they like about recess? How are they feeling?
- 3) After recess, talk about these things that created positive emotion, and how easy it was to pay attention to the good stuff!!!



## Positive Emotion

***Purpose***

To provide students with different opportunities to experience positive emotion, and discover how easy it can be! By the end of this unit, everyone will have a basic understanding of what positive emotion is and how it can benefit their heart health!

***Student Involvement***

Participating students will be involved in a variety of activities and discussions about positive emotion



## Everybody's IT Tag (Knee tag)

Purpose: To discover how easy it is to experience positive emotion! This is just one game that anyone can play on their own, and have fun!

Time: 10 minutes (or more!)

### Procedure:

- 1) Using the same rules as regular tag, *everyone* is it!
- 2) Once you are touched (below the knee) you must sit down
- 3) However, keep track of the person that touched you!
- 4) When the person that touched you gets tagged, you get to stand up and start playing again!!!



## One-Word Story Train

Purpose: To write an outrageous story and be silly! You'll be sure to have a good laugh with this activity!!

Time: 15 minutes

### Procedure:

- 1) On a big piece of flip-chart paper, have each student write down one word at a time, one after another, to create a funny story
- 2) Pass the marker around until the page is filled up or the story is done
- 3) Enjoy the laughter! :)



### Balloon Press

Purpose: Just like your Happy Habits, when your balloon of happiness pops (and your day isn't going so well), just get a new one and start over again — it's that easy! This activity creates a positive atmosphere and works on teamwork, all leading to positive emotion!

Time: 15-20 minutes



Procedure:

- 1) Divide the group in two. Each group needs a team captain. All the other students in the group need to find a partner
- 2) With a partner, each pair will blow up a balloon-- not totally inflated is better
- 3) Place the balloon between your backs and move to the designated area (and back!) without dropping your balloon!
- 4) If you drop the balloon, the captain can place it back for you. If you pop the balloon, you have to get a new balloon, blow it up and start back at the beginning

### Mystery Bag Relay

Purpose: To work on teamwork, cooperation, communication, have a good laugh, and look silly!!!

Needed: silly clothes to go in the mystery bag

Time: 10 minutes

Procedure:

- 1) Place funny clothing in bags, using one bag per team *\*make sure there are enough articles of clothing for each person playing! Extra's are even better!!!*
- 2) Depending on the size of your group, split up into groups of 7-10 people per team
- 3) Place the bags of mystery items up high, about 15m away, so the relay person cannot see what they are grabbing
- 4) On the word "GO" the first person runs to the bag and must dress in whatever item of clothing they select
- 5) The relay runner must fully dress in the item, then go back and tag the next person in their group
- 6) The contest ends when the mystery bag is empty (*again, make sure each team has the same number of pieces of clothing!*)
- 7) Be sure to HAVE FUN!!!

## Gotcha

Purpose: To get the whole group laughing!!! Time: 5-10 minutes

Procedure:

- 1) Have the group stand in a circle around your leader
- 2) Each person puts their left hand out flat to their left side, and their right index finger on the person's hand that is beside them
- 3) The finger should be slightly above the palm of the hand, but not touching, so the person can squeeze it
- 4) Have your leader tell a funny story, and whenever a predetermined word is said, all the left hands try and grab their neighbours' index finger
- 5) The object is to escape the person to your right and catch the person to your left!
- 6) The leader can switch it up and use different trigger words, or even a number, and start counting in different sequences
- 7) This game usually leaves everyone smiling, if not laughing!!



## Negative to Positive Shifts

Purpose: To learn how to shift negative attitudes to be more positive! It's just like they say — turn that frown upside-down!

Time: 15 minutes

Procedure:

- 1) Give each student a negative statement
- 2) Ask them to rewrite the statement so that it becomes positive!

Example Statements:

Nobody likes me      I am dumb

I am ugly              I am fat

I am not important   I never do anything right

My grades are awful



Purpose: To show that people perform acts of kindness all over the world!!!

Time: 20 - 30mins

Procedure:

- 1) Have students go through newspapers, searching for acts of kindness, and cutting them out
- 2) Create a collage or poster of these acts
- 3) If there is time, go around in a circle and share these acts with the group
- 4) Be sure to hang these acts of kindness somewhere for everyone to see! *(\*if needed, highlight the kindness-related parts of the article\*)*

Positive Emotion

*Gratitude & Kindness*

***Purpose***

To provide students with different opportunities to experience positive emotion, and discover how easy it can be!

By the end of this unit, everyone will have a basic understanding of what positive emotion is and how it can benefit their heart health!

***Student Involvement***

Participating students will be involved in a variety of activities and discussions about positive emotion.

## Positive Affirmations

Purpose: To be able to identify and create positive statements, and to learn how to turn a negative into a motivating, positive statement! Positive affirmations help reduce stress and increase a feeling of personal power.

Time: 20-25 minutes

Procedure:

- 1) Ask the group to name some examples of positive thoughts
- 2) Ask the group to list some negative self talk
- 3) Why is it important to think positive thoughts?
- 4) Give some examples of positive affirmations

- Challenges help me grow
- I can see stressful situations as challenges
- Challenges bring opportunity
- I can choose a positive frame of mind

5) Have the group create their own positive affirmation, and turn it into a poster to be shared with everyone!

6) If there is time, talk about your statement with the group, and hang the posters around school



## Random Act of Kindness Club

Purpose: To show your school all the different acts of kindness that people perform, and to spread awareness, showing that acts of kindness can be easy!

Time: ongoing (10 minutes to explain) Procedure:

- 1) Explain to your group that people do random acts of kindness every day!
- 2) Once a 'club' member has done an act of kindness, ask them to make a poster about that act, and post it on the wall (*\*could be added to the Kindness Path\**)
- 3) Make a group goal, such as asking each person to perform at least 1 act of kindness a day



## Alphabet List

Purpose: To create a huge list of acts of kindness! Everyone knows the alphabet, so show them how easy it is to do an act of kindness - it can be anything, big or small! This activity will help raise awareness about doing acts of kindness, and encourage others to do the same!

Time: 10 - 25 minutes

### Procedure:

- 1) Distribute the letters of the alphabet among the group. Ask your students to come up with an act of kindness that starts with that letter eg. Aided a younger kid on the playground  
Bought a milk for a grade 1 student Caught a ball over the fence for a team
- 2) Write the act of kindness on the letter, and hang the alphabet in a visible area



## P.A.L. Project

Purpose: To help find the good in anyone and everyone!!! Time: 10 minutes

### Procedure:

- 1) Put all the participants names in a hat
- 2) Each student draws a name, but SHHHH, don't tell who's name you picked!!!
- 3) Over the next week, write or type a letter about the person who's name you picked. This letter should include things like why they are a good person, things they do well, things you could encourage them on, etc., etc.
- 4) The letter should say "To: <student name>, From: Anonymous"
- 5) After your leader looks over the letter, hand back the letters next week, and read all the kind things people have written about you!



## First Impressions



Purpose: To show that you have more in common with your peers than you think! This also shows that each of you are unique and have something different to contribute to the group.

Time: 15-20 minutes

Procedure:

- 1) Pass out large sheets of paper and markers
- 2) Have each person write their name on the top of the paper, and tape it to their back
- 3) Ask everyone to mingle. Ask them to say hello and introduce themselves for a few moments. After a minute or so, ask each person to write an adjective - their "first impression" of the person they just spoke with, or a word that describes them- on each other's papers

*\*\*please note: write encouraging words ONLY!!!*

- 4) Next, continue mingling with new people, repeating the process
- 5) After 10mins, each person should have several words listed on their backs
- 6) Going around the room, introduce each other and read the words written on your neighbour's paper

## Angel Box



Purpose: To recognize acts of kindness that *others* do, and help them realize they make a difference, and their kindness is noticed!

Time: ongoing (10 minutes to explain)

Procedure:

- 1) Ask students to watch for acts of kindness throughout the week
- 2) When you see an act of kindness, write it down on a piece of paper and put it in the drop-box. Be sure to include the name of the person that you saw being kind!
- 3) Let the students know that these acts of kindness will be read to the group the following week. *As a challenge, don't read out the persons' name, and see if they can recall their action!*

### Walk the Path to Kindness

Purpose: To create a path of kind acts, inspiring your classmates to do the same! All of this will help lead to a healthier, happier life!

Time: ongoing (10 minutes to explain) Procedure:

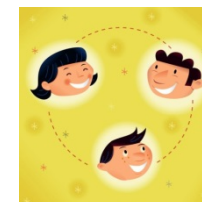
- 1) Talk to the students about how committing acts of kindness helps lead to a happier, healthier life, and discuss why (try asking your classmates why they think it is important, rather than telling them!)
- 2) Pick a location on a wall that has room for a pathway. Set up some pieces of paper to write on, pens, and tape, and be sure to include brief instructions
- 3) Tell the students to write their act(s) of kindness on a cut out, and make a pathway, all leading to a happy sun or smiley face, sharing the path for others to see!



### Positive Emotion of the Day!

Purpose: To melt away your morning tension, and turn your day around - into a day full of positive emotion!

Time: 10 - 15 minutes



Procedure:

- 1) Sit in a circle
- 2) Go around the circle and have your peers say one good thing that the person next to them has done in the past 24hrs (eg. opened the door for you, been a good friend, been a good listener, made you laugh, was a team player, etc.)
- 3) Ask all the other students to raise their hands if they agree
- 4) Encourage your peers not to repeat a trait that someone else has used

### Sit Spot

Purpose: To help you learn how to use a favourite spot (hopefully outdoors) to just sit and chill and help you cope, letting all the bad vibes out, and the positive emotion in!

Time: 15-20 minutes

Procedure:

- 1) Inform your peers that you will be heading outdoors. Ask them to do the following:  
*[\*\*note: try to stay away from the playground!!]*
  - find a place in the outdoors where you feel comfortable
  - listen carefully to the different sounds you hear for 10 minutes
  - look around and experience what you see going on around you
  - use all your senses while sitting in your Sit Spot
- 2) Ask your peers to answer these specific questions, and consider their emotions:
  - What did you hear, see, smell, feel?*
  - What did you like most about your sit spot?*
  - Why did you pick this particular spot?*

\*Ask any other questions you find appropriate

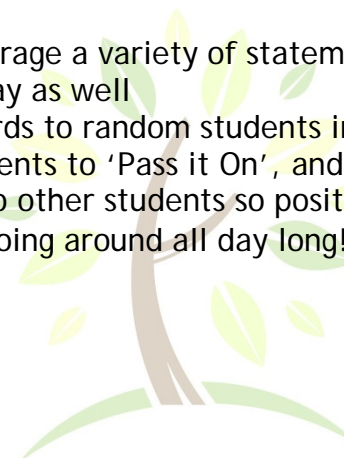
### Pay it Forward

Purpose: To send happy, uplifting, positive messages to your student population, putting a smile on at least one other person's face!!!

Time: 15 minutes

Procedure:

- 1) Pass out paper/cue cards to each student
- 2) Ask students to write positive statements such as: Your smile makes me smile! You are a champion! You are electric! Decorate with smileys and symbols
- 3) Write 'Pass it On!' on the flip side of the paper
- 4) Try to encourage a variety of statements, but doubles are okay as well
- 5) Hand out cards to random students in the hall
- 6) Remind students to 'Pass it On', and continue passing cards to other students so positive messages are going around all day long! :)



## Mirror of Strengths

Purpose: To peel away the layers and find out what strengths others see in you - sometimes you can't always see your own strengths, but they are what many admire most!

Time: 15-20 minutes

Procedure:

- 1) Ask the group to find a partner (you can do this multiple times, finding new partners each time. Try sorting by birth month, eye colour, etc.) and spread out
- 2) Ask one person to act out the strengths they see in their partner, essentially *mirroring* them, and have the partner guess that strength
- 3) Afterwards, discuss each person's strengths within the pair. If there is time, share these discoveries with the group

Debrief:

- 1) What did you learn about yourself today?
- 2) Why is learning your strengths important?
- 3) How can you use these strengths to generate more positive emotion in your life?

## Team Resumé

Purpose: To focus on collective strengths within the group, and is a great way for students to learn new things about their peers. You'll be amazed at how much your group has to offer!!!

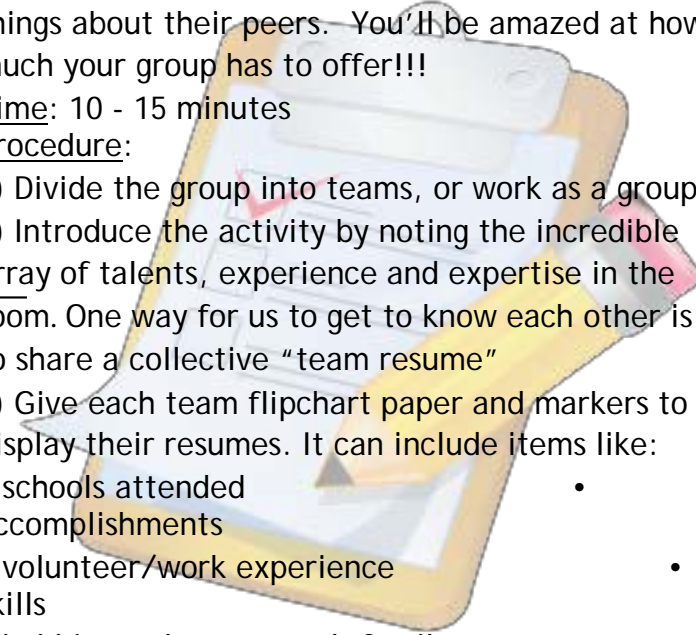
Time: 10 - 15 minutes

Procedure:

- 1) Divide the group into teams, or work as a group
- 2) Introduce the activity by noting the incredible array of talents, experience and expertise in the room. One way for us to get to know each other is to share a collective "team resumé"
- 3) Give each team flipchart paper and markers to display their resumes. It can include items like:
  - schools attended
  - accomplishments
  - volunteer/work experience
  - skills
  - hobbies, talents, travel, family
- 4) Encourage your peers not to repeat a trait that someone else has used

Debrief:

- 1) What was today's activity about, and why is it important?
- 2) What have you learned about our group, and how does this impact everyday life?



## What Would Your Superhero Look Like?

Purpose: To showcase your strengths on your own superhero character, emphasizing what you are best at for all to see!

Time: 15-20 minutes

Procedure:

- 1) Pass out large sheets of paper and markers
- 2) Have each person write their superhero name on the top of the paper, and draw their personal superhero character
- 3) Remember to include your strongest strengths, plus a few favourites (ie. fave team jersey, etc)!
- 4) After approx. 15mins, each person should have a character created - going around the room, introduce your superhero and highlight the strengths it possesses

Debrief:

- 1) What was today's activity about and why is it important?
- 2) How do you see yourself using these superhero strengths in your everyday life?

## Body Map

Purpose: To recognize your own interests and talents, and how they relate to others. This activity will help you learn about each other's differences and strengths, and help you share skills together!

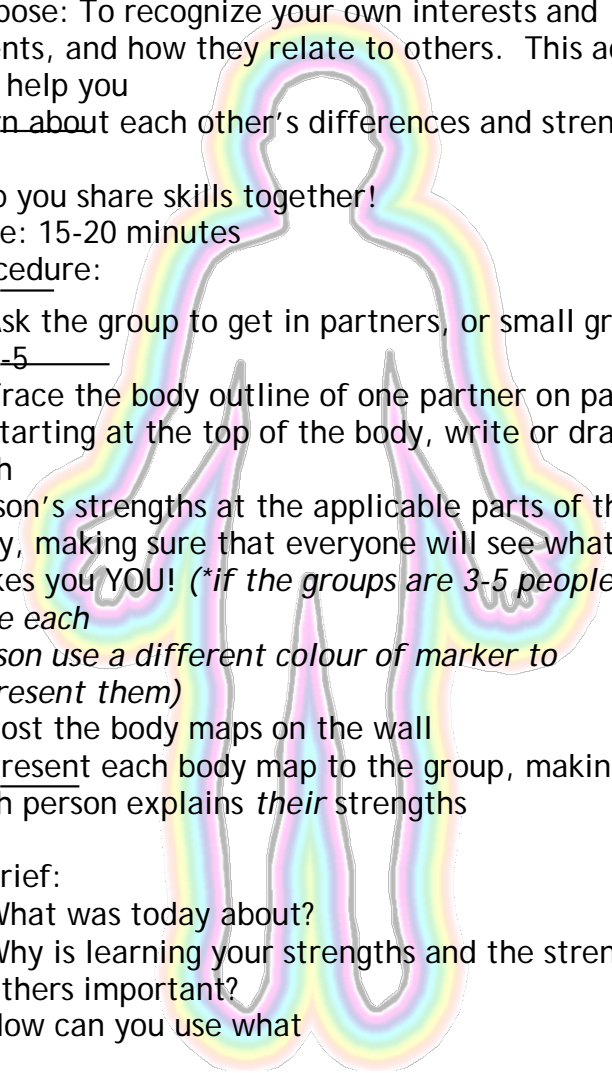
Time: 15-20 minutes

Procedure:

- 1) Ask the group to get in partners, or small groups of 3-5
- 2) Trace the body outline of one partner on paper
- 3) Starting at the top of the body, write or draw each person's strengths at the applicable parts of the body, making sure that everyone will see what makes you YOU! (*\*if the groups are 3-5 people, have each person use a different colour of marker to represent them*)
- 4) Post the body maps on the wall
- 5) Present each body map to the group, making sure each person explains *their* strengths

Debrief:

- 1) What was today about?
- 2) Why is learning your strengths and the strengths of others important?
- 3) How can you use what



## String of Strengths

Purpose: By understanding your strengths, you will have a better understanding of what sort of person you are. Being able to surround yourself with and use these positive strengths is a good thing, so let's get started!

Time: 20 minutes

### Procedure:

- 1) Label a cut-out of a person to represent you
- 2) Surrounding this paper cut-out, add a circle of string. Using the different shape cut-outs, write a different strength on each shape, and stick the shape to the string. Try to come up with at least 10 different strengths
- 3) The end result will be a person (YOU!) surrounded by unique and individual strengths that you can use throughout your daily life. Be sure to share these great qualities with the group!

### Debrief:

- 1) Why is it important to remind yourself of your strengths?
- 2) Why is it important to use these strengths in your everyday life?
- 3) How else can you use these strengths in your daily life?



### What Animal Are You?

Purpose: To help students recognize their strengths, and what type of person they are - whether they are more controlling, promoting, supporting, or analyzing.

Remember, each trait is important for different reasons!!

Time: 15 minutes

### Procedure:

- 1) Explain to the group that everyone has different strengths that they carry, and no strength is more important than another
- 2) When you add up all your strengths, you tend to have certain personal traits that can be likened to a lion, otter, golden retriever, or beaver. Ask the students to complete the questionnaire in order to determine what animal they are most similar to
- 3) Once the results are totaled, students will have a better idea of their personal traits, and how they tend to work with others
- 4) If there is time, share your results with the group, pointing out that it is important to have a mix of styles in groups!



### Debrief:

- 1) Why is it important to be able to recognize your strengths?
- 2) What have you learned about your strengths?
- 3) How do you see yourself using these strengths in your everyday life?



## Multiple Intelligences Questionnaire

Purpose: To help students learn their strengths and recognize that everyone has strengths in each area, some are just stronger than others! This questionnaire can help to empower students (*\*not label them!*)

Time: 20-25 minutes

Procedure:

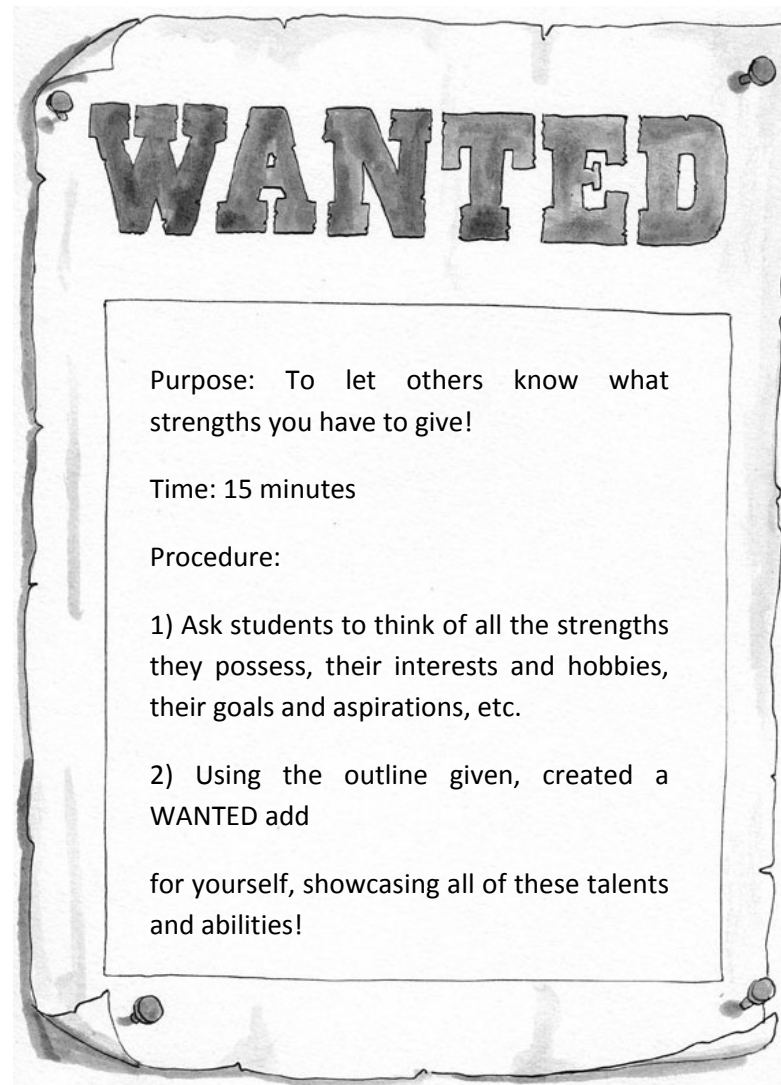
- 1) Explain to the group that everyone has different strengths that they carry, and no strength is more important than another
- 2) Ask the students to complete the questionnaire in order to determine the areas they are strongest in
- 3) Once the results are totaled, students will have a better idea of where their strengths lie!
- 4) If there is time, share your results with the group. Remember to point out that each area of strength is important for different reasons, and having a mix of strengths within a group is very positive!

Debrief:

- 1) Why is it important to learn your strengths?
- 2) What have you learned about your strengths?
- 3) How do you see yourself using these strengths?

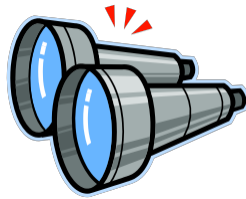








## Leisure Scope



Purpose: To help students discover what activity categories interest them and feel natural. Check out some cool images of sports, nature, collection, arts, music, education, entertainment, and culture, volunteering, and organization, all to help you discover what interests you and why!

Time: 15-20 minutes

Procedure:

- 1) After reminding the group that everyone has different strengths, and they are all equal, pass around the collages of the themes listed above
- 2) Ask the students to think about which 3 collages represent their highest level of interest
- 3) After ranking their fave themes, pass around the collages again, this time thinking about what feelings come up regarding those images (eg. relaxation, health, excitement, relief, fun, escape, pleasure, satisfaction, etc)

Debrief:

- 1) What did you learn about yourself today?
- 2) Why is learning about your leisure choices, and how they make you feel, important?
- 3) What does all of this mean? How can you use it?



Strengths + Free Time Activities



### *Purpose*

To provide students with different opportunities to discover and experience their personal strengths. By the end of this unit, everyone will have a basic understanding of what their strengths are, how to use them, why it's important to use them, and how it can benefit their heart health!

### *Student Involvement*

Participating students will be involved in a variety of activities and discussions about personal strengths.

## What Am I?

Purpose: To show students various activities that require different strengths, and perhaps they would like to try some of them! By identifying leisure activities, students can brainstorm the strengths needed in each activity

Time: 20 minutes

Procedure:

- 1) Have small pieces of paper with activities written on them
- 2) Ask everyone in the group to find a partner. Give an activity card to one person in each group *\*don't show your partner the card!!\**
- 3) Ask the person to act out the activity on the card while the partner tries to guess the activity
- 4) Once they have guessed the leisure activity, brainstorm what strengths are required to perform that activity
- 5) Share your activity and required strengths with the group

Debrief:

- 1) Why is it important to know what activities use your strengths?
- 2) How do you see yourself choosing activities that use your strengths?

## Superhero Actions

Purpose: To give your superhero strengths some activities to rock! Be zany, be wacky, be creative - pick some activities that best fit your superhero strengths!

Time: 10 - 15 minutes

Procedure:

- 1) Using your superhero that you created last week, think about the strengths it possesses (and ultimately *YOU* possess)
- 2) Focusing on those strengths, create a list of at least 15 activities that would suit your superhero, and centre on your shared strengths
- 3) Present your superhero and it's strengths to the group. Be sure to share the leisure activities you have highlighted to go with each strength!

Debrief:

- 1) What did you learn from this activity today?
- 2) Why is it important to know what activities suit your strengths the best?
- 3) How can this knowledge impact your everyday life?



Purpose: To seek out strengths and identify leisure activities that develop or use that strength.

Get your searching skills ready to have fun!!

Time: 25-30 minutes

Procedure:

- 1) Write a list of strengths on colourful pieces of paper - *be creative!*
  - 2) Pick locations around the school to hide these strengths and write clues that direct the students to the hidden strengths
  - 3) Don't forget to hide the clues *with* the strengths!  
*\*tip: colour-code and number (or letter) the clues so that all the teams aren't going in the same direction*
  - 4) Divide the students in to small groups. Outline how a scavenger hunt works, and explain that they need to come up with leisure activities that develop or use the strength they find
  - 5) Hand out the first clue to each team, and off they go! Be sure to walk around and offer help!
  - 6) Regroup and ask what leisure activities they would take part in to develop or use that strength
- Debrief: 1) What was today's activity about and why is it important?
- 2) How do you see yourself choosing activities that use your strengths?

## Leisure Interest Measure (LIM)

Purpose: To learn about the types of leisure activities you prefer, or want to do, all based on your strengths!

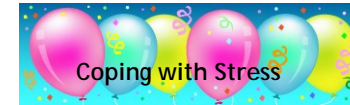
Time: 15 minutes

Procedure:

- 1) Remind the students that everyone has different, equally important strengths. To get the most out of these strengths, there are different activities that you might like to do, or want to try
- 2) There are 24 statements on the LIM. Using the numbers 1 (never true) - 5 (always true), indicate how true each statement is. Remember, there are no right or wrong answers!!
- 3) Use the scoring system to determine what types of leisure interests are most suited to you (reading, outdoor, mechanical, cultural, service, physical, social, artistic)

Debrief:

- 1) What did you learn from this activity today?
- 2) Why is it important to know what activities suit you best?
- 3) How do you see yourself using this information in your everyday life?



## Zen Breathing

Purpose: To briefly escape from your day-to-day stresses and worries using a relaxation coping strategy

Time: 15 minutes

Procedure:

- 1) Sit in a comfortable position
- 2) Close your eyes, keep your back straight, shoulders relaxed, head up, focused ahead
- 3) Take a deep, cleansing breath, expanding your belly and keeping your shoulders relaxed
- 4) Hold this for 6 seconds. Exhale, and repeat twice more
- 5) Then breathe normally, and focus your attention on your breathing. As you breathe, inhale through your nose and exhale through your mouth, still expanding your belly rather than moving your shoulders up and down

*\*If your thoughts drift to the stresses of the day ahead or of the day behind you, gently refocus on your breathing and remain in the present moment. Feel the air move in, and feel the air move out*

Debrief:

- 1) How did this make you feel? Were you more relaxed following this activity?
- 2) Would you like to continue to use this to close the club?
- 3) Would you use this at home when you start to feel overwhelmed?
- 4) When do you feel most stressed? What time of the day, in what situation?

Purpose: To identify stressors and symptoms, learn the importance of managing personal stress, examine ways to manage stress, and create a plan of action to manage stress

Time: 30 minutes

Procedure:

- 1) Discuss what stress is. Ask students about some of the stressors in their life and write them down on chart paper
- 2) Introduce Balloon-Human Analogy - the balloon does not have any air in it and therefore, it is deflated. Adding air to the balloon creates some energy, it regulates stimulation
- 3) Start blowing the balloon up; as you blow it up, the more stressed the balloon gets
- 4) Start mentioning stressors from the list of stressors that are written on the chart. *"What will happen if I continue to pile stressors (air) into this balloon?"*
- 5) Mention a few more stressors and then pop the balloon with a sharp object
- 6) Explain the difference between Eustress (*good stress= a little bit of the air in the balloon that stimulated excitement and good energy*) and Distress (*too much air= too much piled on stress*)
- 7) Hand out a 'stress symptoms' handout sheet. Ask the students to check off any box that fits the emotion(s) they feel when stressed
- 8) Ask *why it's important to manage stress; what can uncontrolled stress lead to and what does it affect?*

Ask students to fill out handout #2: Positive Changes. Ask them to *develop a plan to deal with the stressors in their life*

Debrief: occurs throughout the procedure (questions are being asked to prompt reflection)

### Keypunch

Purpose: To learn how to work together, communicate effectively, and improve performance by managing your group stress!

Time: 30 minutes

Procedure:

- 1) Place objects numbered 1-20 in a circle made from rope (or on a table, etc). The group stands "on base" in a different area, 15-20ft from the circle
- 2) Explain that the group must start and finish on base. Once you hear "Go", the group must run to the calculator and take turns pressing the numbers 1-20 in order. Once they have reached 20, they must return to base. The timer starts when they leave base and stops when the whole group has returned. The goal is to do this as fast as possible
- 3) Give students 5mins to plan, and then start the timer for the first attempt
- 4) After the first attempt, explain that the students have 20mins to discuss and better their time, taking as many attempts as needed
- 5) The rules are as follows:
  - a) group must start and finish on base
  - b) everyone in the group must touch a #
  - c) only 1 person can reach into the circle at a time
  - d) everyone must remain on the outside of the circle - no stepping across or standing inside
  - e) if a # is touched out of sequence, or rule c or d are broken, the group must start over

Debrief:

- 1) What did the stress feel like as time got closer?
- 2) How did you (and the group) deal with it? What other strategies could you have used?
- 3) How did your body react? What are 2 things you do that make your body feel the same way?

### Stress Circle

Purpose: To help students discover what they are most stressed about, & what makes them & their peers stressed

Time: 15 minutes

Procedure:

- 1) Have statements prepared that you think might stress a fellow student (eg. night before a test, etc)
- 2) Create a small circle (big enough so that 10 people could fit tightly inside) and a larger circle on the outside of the smaller one (with enough space so that people could fit in-between them comfortably), similar to a target
- 3) The inner circle represents not stressed, between the two circles represents a little stressed but still manageable, and the outer biggest circle represents high stress/anxiety and not manageable
- 4) Say one stressor to the group at a time
- 5) Let kids decide how stressed they would be by going to the part of the circle that best represents how they would feel in that situation

Debrief:

- 1) What did you find was your biggest stressor?
- 2) What can you do to help reduce negative stress in your life?

## Restful Place

Purpose: To provide students with an example of an activity that can help them cope with stress, and decrease their stress level

Time: 15 minutes

Procedure:

- 1) Ask the students what helps them relax
  - a) how can music help you focus on positive things and take your mind to a restful place?
- 2) Tell students to close their eyes, lie in a comfortable position and think of their favorite location
- 3) Turn on music in the background and listen to music for a few minutes while relaxing

Debrief:

- 1) How did you feel during the 10 minutes?
- 2) When would this strategy be effective in helping you?
- 3) How does this activity decrease your stress level?
- 4) Discuss with a partner how spending a few minutes each day in rest could help focus their mind and decrease stress



## Traffic Jam

Purpose: To increase problem solving, teamwork, communication and coping skills

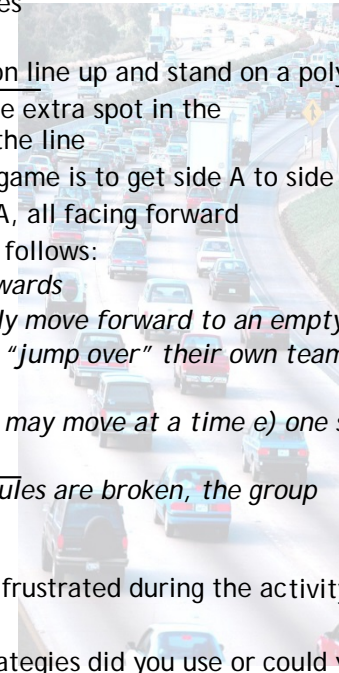
Time: 20-30 minutes

Procedure:

- 1) Have each person line up and stand on a poly spot (carpet square), placing the extra spot in the middle of the line
- 2) The goal of the game is to get side A to side B and side B to side A, all facing forward
- 3) The rules are as follows:
  - a) *no moving backwards*
  - b) *a person can only move forward to an empty space*
  - c) *a person cannot "jump over" their own team mate*
  - d) *only one person may move at a time*
  - e) *one spot per person, no sharing*
  - f) *if any of these rules are broken, the group must begin again*

Debrief:

- 1) Did anyone feel frustrated during the activity? How did you deal with this?
- 2) What coping strategies did you use or could you have used to help deal with this?
- 3) What were the sources of stress in this activity?
  - a) what comes from outside?
  - b) what comes from inside?
- c) how did you deal/respond to it? Was it different from the outside vs. inside stress? How?
- 4) In what ways was the stress helpful/not helpful?







Purpose: To allow students to use the coping strategies they have learned to act out different stressful situations

Time: 30 minutes

Procedure:

- 1) Students will get into groups of 4-6
- 2) Ask them to choose from a list of scenarios
- 3) Give them 15mins to come up with a 2-5min skit
- 4) Ask each group to act out the skit in one of two ways, either: without coping strategies, with coping strategies

*\*Example scenarios: preparing for a big test, argument with friend(s) or sibling(s), parents having problems, not having enough privacy, birth of a brother or sister, moving to a new school, a teacher who doesn't like you*

Debrief:

- 1) Which coping strategies were used in the skits? Why?
- 2) What are the kinds of things that stress you out?
- 3) What have you learned that might help you cope in the future?

## Coping Scramble

Purpose: To teach students about the types of coping strategies

Time: 10 - 15 minutes

Procedure:

- 1) Have participants stand in the centre of the gym
- 2) Identify each wall as a type of coping strategy (emotional, problem, distraction/avoidance, relaxation)
- 3) The leader calls out a statement and participants run to a wall identifying what type of strategy it is

*\*Example statements: talking to a friend, listening to music, reading a book, going for a walk, writing study notes, asking for help, ignoring the problem, meditation, making a priority list*

Debrief:

- 1) Define what the different types of coping are
- 2) Identify what coping strategies you use
- 3) What are some other strategies that you think could also work for you?





### Free time and Vitality: It's Your Choice!

**Introduction:** When we think about creating a healthier life, one place that we have the most control is in our free time. We are going to think a bit about how you spend your time, and talk a bit about how to make great choices with your time so that your free time helps you develop your strengths and helps you feel more positive about your life.

When you think about free time, there are a number of reasons why free time choices are so important for your overall health and well-being. Those times in your life when you are not expected to be doing other things give you a chance to make decisions about where you are going to invest your energy and attention. Free time is a time when you can make choices that help you come to know yourself better; you can experiment with different activities that help you figure out what your strengths are and what makes you happy. Your free time is a time that you can use to create more balance in your life by choosing active pursuits if you have spent all day in school, or by choosing relaxation activities if you are feeling stressed. Finally, free time is a great time to purposefully seek out experiences that generate positive emotion and happiness.

#### Free time Patterns

- 1) Review of weekly obligated activities (school, homework), free time (recreation, relaxation, socializing), and mixed (organized sports, music lessons, etc.)
- 2) Benefits of making free time choices
- 3) Connection between free time choices and positive emotion, strengths and coping

#### Free time Patterns:

*Goal:* Identify the amount of free time you have available in a typical week.

*Goal:* Identify two reasons that free time choices are important for your health and well-being.

*Goal:* Identify the connection between free time choices, strengths, positive emotion, and coping.



### Free Time Choices

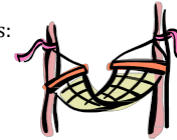
1) Types of free time choices:

- |                             |   |
|-----------------------------|---|
| a) Creative/self-expressive | e) Hobbies/crafts/expressive arts                 |
| b) Physically active        | f) Civic engagement/volunteering                  |
| c) Socially active          | g) Electronics (TV, video games, internet, music) |
| d) Spectatorship            | h) Music (playing an instrument, etc.)            |



2) Free time activities are usually experienced in contrast with other activities:

- a) Relaxation vs. obligated activities  
b) Social vs. solitary  
c) Physically active vs. sedentary



3) Flow zone activities engage us and help us develop skills. Selecting those activities involve:

- a) Finding activities that allow for levels of performance  
b) Finding activities that are interesting and engaging  
c) Finding activities that allow us to become so involved that we lose track of time



#### Free time Choices:

*Goal:* Identify three types of free time activities that are of interest to you.

*Goal:* Identify one activity that gets you into the flow zone.

*Goal:* Identify two things to consider when selecting activities that help you get into the flow zone.

